Journal

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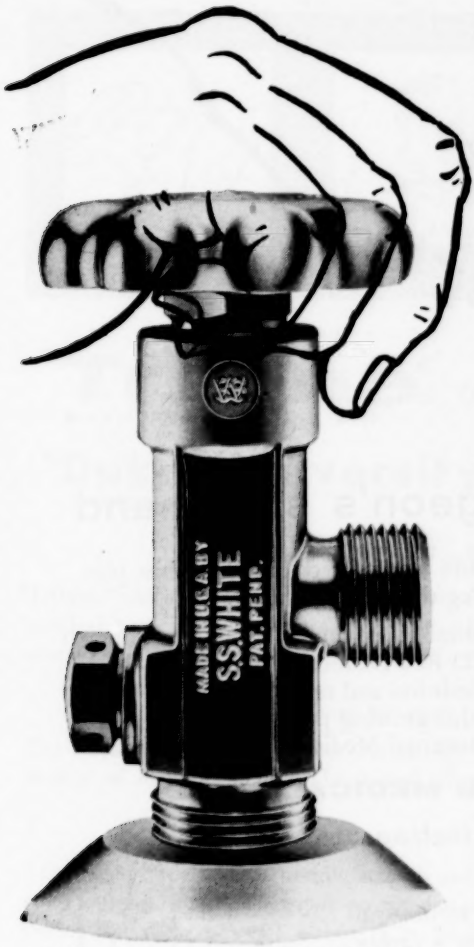

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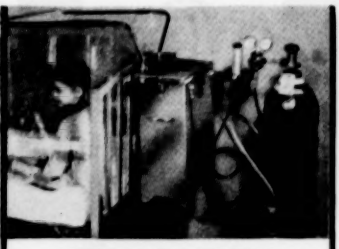

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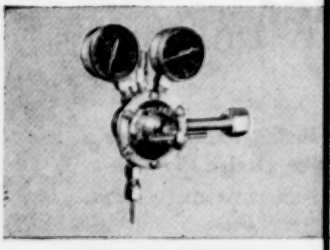

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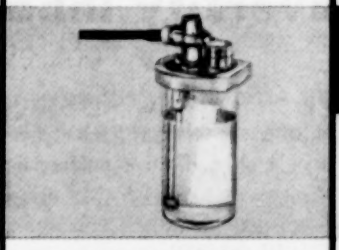

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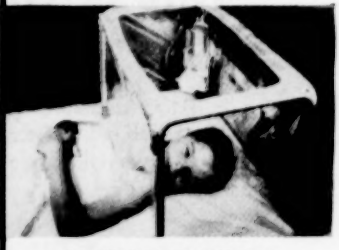

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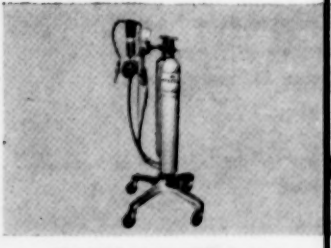

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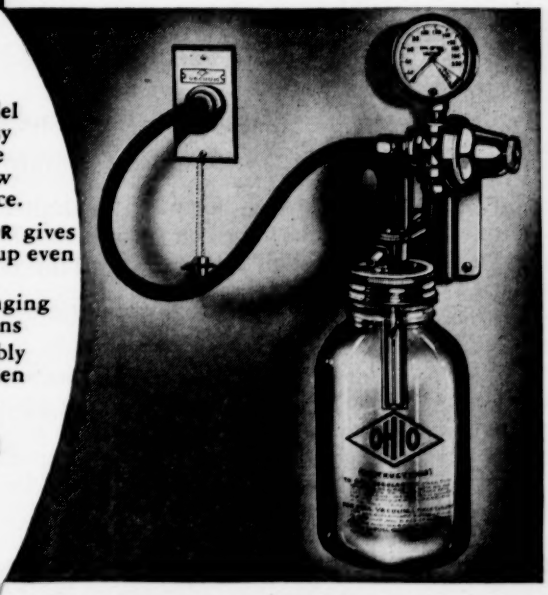

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Survey of Anesthesia Service: 1955

Mrs. Jessie L. Compton, R.N., Marie N. Bader, R.N., Minnie V. Haas, R.N., and Agnes M. Lange R.N. *

In conducting its programs and in planning for the future, the Board of Trustees of the American Association of Nurse Anesthetists has long been aware of the need for an accurate estimate of the number of persons who administer anesthesia in the United States. In 1950, a survey was made by sending a questionnaire to members of the A.A.N.A. From an analysis of that questionnaire it was estimated that 16,815 anesthetists were needed and that there were at that time only 5,515 persons with known qualifications as anesthetists. Since that questionnaire was sent to members of A.A.N.A. only, it was recognized "that all these hospitals had qualified anesthetists in their employ, either as full-time or part-time workers, and the statistics should not be interpreted as being representative of all hospitals."[1]

It is the belief of this association that opinions and guessing do not supply an adequate basis for estimating the present supply of persons in the field and cannot serve as a basis for improving the situation.

Since the 1950 survey did not cover all hospitals, and since the data are probably obsolete because of the rapid changes in the hospital field, and because the shortage of anesthetists still exists, a new survey was indicated. To determine the present situation, a questionnaire was sent to all hospital administrators in the United States and its territories and possessions. In requesting participation in the survey the following statement was made: "Before a realistic approach could be made to solving the problem of the apparent continuing shortage of anesthetists it would seem necessary to know who is now administering anesthesia." The hospital administrators were asked to supply the following information: "During the month of January 1955 what persons were giving anesthesia in your hospital; the name and title (R.N., M.D., etc.); the approximate number or percent of anesthesias given by each. If anesthesia service in the obstetric department is not covered by the persons listed please list the names separately."

The Response

7,032 questionnaires were mailed; 3,235 were returned. Of the 3,235 that were returned 13% were from hospitals that did not have anesthesia departments or did not supply the information requested in a manner that could be used. For instance, among those that did not supply

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*Members of the Planning Committee.

Presented at the Business Session of the American Association of Nurse Anesthetists meeting in Atlantic City, September 20, 1955.

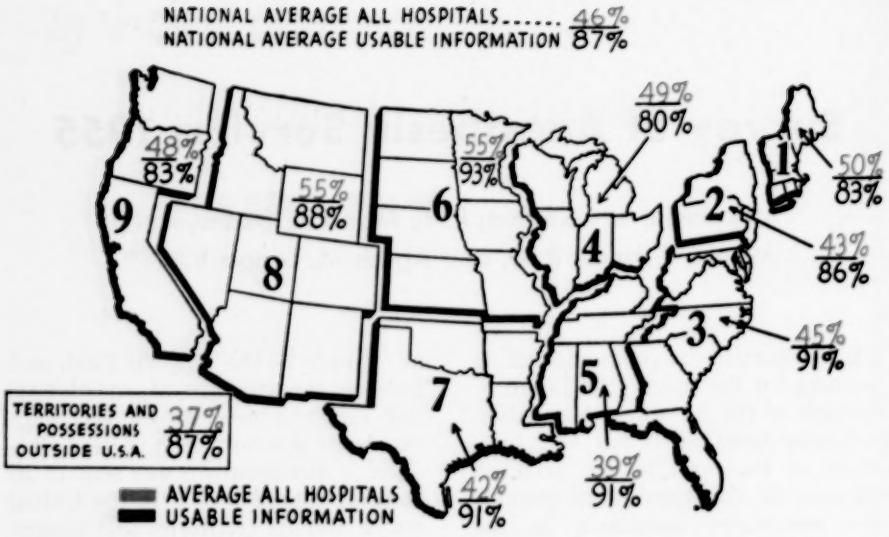

Figure 1. Percent of questionnaires returned from hospitals in geographic regions and the percent of usable returns.

usable information some of the reasons given were:

"We do not furnish names of our personnel to outside agencies

"This hospital has one physician anesthetist and 5 nurses (no names being given)

"All anesthesia is administered by physicians in general practice, a very unsatisfactory situation that we hope to change and one that I do not like

"We have only registered nurse anesthetists. . .we are very happy with this service

"The law. . .in this state does not provide for anesthesia to be given by nurses

"We formerly employed nurse anesthetists but have been unable to get such a person

"No anesthesia given; reason—surgery was discontinued because of the lack of an anesthetist"

The Validity of the Response*

The questionnaires that were returned were tested to evaluate whether they represented an adequate coverage both geographically and by hospital size. They were divided according to four sizes of hospitals: 0 to 49 beds, 50 to 99 beds, 100 to 249 beds, 250 beds and over, and were then sorted according to state. Information for hospital size was obtained from the 1954 Hospital Administrators Guide published by the American Hospital Association. [2]

To test whether there was significance in the location or the size of

*The statistical analysis of the current survey was computed by the Counselling and Testing Service, Roosevelt University of Chicago, Illinois, Alyce E. Graham and Richard E. Pasca, Co-Directors. The professional staff members of the Counseling and Testing Service have been Test Construction Consultants for the A.A.N.A. since 1952. They have been primarily concerned with the construction, scoring and item analysis of the Association's Qualifying Examinations.

the hospitals that did not return the questionnaire, these were counted in the AHA Directory and compared by size and location with those hospitals that did return questionnaires. There did not seem to be a statistical difference in the replies and non-replies. Figure 1 gives the percentage of replies from each of ten regions, the black number indicating the percent of usable questionnaires from that region, the red figure being the percentage of all hospitals in the region represented in the total return.

Response by hospital size varied; questionnaires having been returned from 39% of the 0 to 49 bed hospitals; 49% of the 50 to 99 bed hospitals; 55% of the 100 to 250 bed hospitals and 51% of the 250 bed and over hospitals.

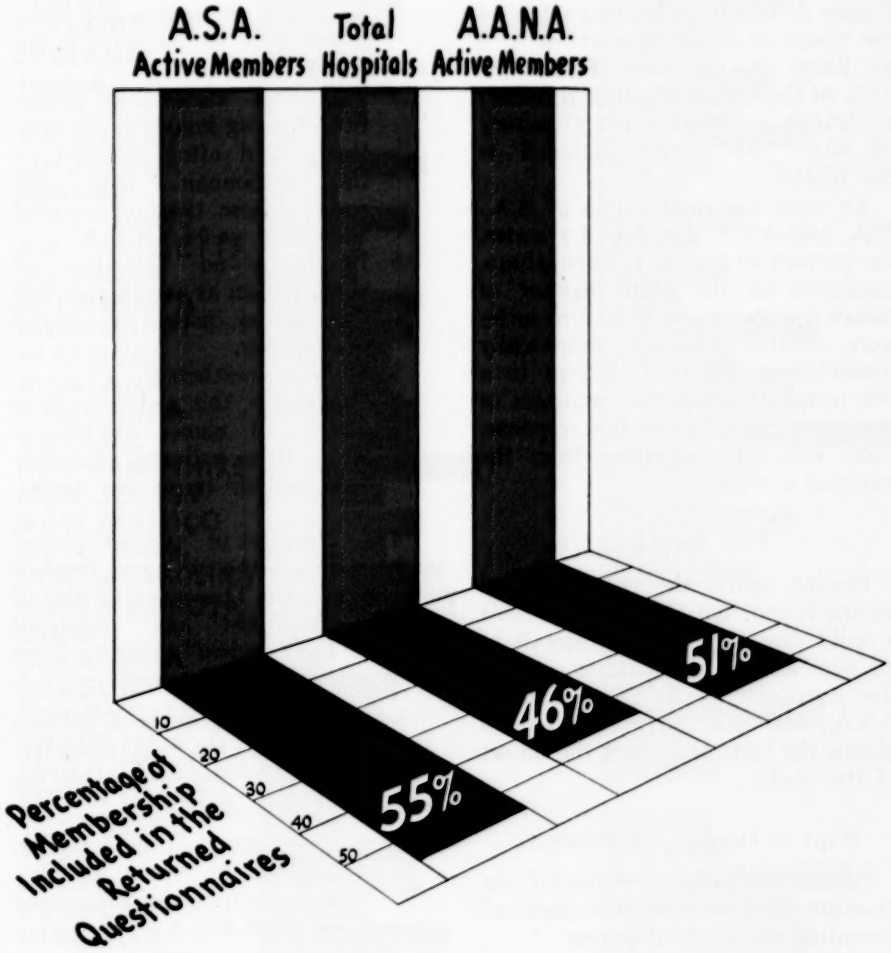

Figure 2. Percent of names of members of AANA and ASA in relation to percent of questionnaires returned.

The national average was a 46% returned questionnaires, 87% of which contained information that was usable in this study.

To further test the coverage of the replies the information was classified according to the percentage of members of the American Society of Anesthesiologists and members of the American Association of Nurse Anesthetists and the percentage compared to the total response. The results of this analysis is illustrated in Figure 2, which shows that 55% of the names of active members of ASA as listed in its 1954 Directory[3], 51% of the names of active members of AANA as listed in its Directory of May 1954[4] were included in the replies.

To show the distribution of A.A.-N.A. and A.S.A. members by states, the percent of names in each group, compared to the total number of names in the respective directories were charted and are graphically presented in Figure 3. Except in a few instances where the smallness of the sampling affected the response, there was little variation from the national averages.

The Analysis

Having tested the responses and having found that they did represent a valid sampling of the hospitals of all sizes and of all locations, and of the members of A.A.N.A. and of A.S.A., the data were analyzed to obtain the facts that were the object of the study.

Who is Giving Anesthesia?

In the preliminary review of the questionnaires persons were classified according to eight categories:

1. Members of the American Association of Nurse Anesthetists as indicated by their listing as active members in the A.A.N.A. Directory.
2. Members of the American Society of Anesthesiologists as indicated by their listing as active members in the A.S.A. Directory.
3. Nurse anesthetists—so designated in the replies, but whose names could not be found in the A.A.N.A. Directory.
4. Physician anesthetist—so designated in the replies but whose names were not included in the A.S.A. Directory.
5. Registered nurses—no indication of being known as an anesthetist and often included on the questionnaires with other names whose titles were listed as R.N.A. or M.A.A.N.A.
6. Physicians—no indication of being known as anesthetists and most often listed as general practitioner.
7. Student anesthetists.
8. Others—in this category were placed all names of persons whose titles indicated that they were neither nurse nor doctor.

The categories of "physician" and "physician anesthetist" were often not clearly defined. This was true also of "nurse anesthetist" and "registered nurse." The "student" category listed both physicians and nurses.

Since the qualifications of persons in these groups could not be determined it was decided to combine the groups into five categories.

1. Active members of the American Association of Nurse Anesthetists, for purposes of the graphs listed as A.A.N.A.
2. Active members of American Society of Anesthesiologists, for

Figure 3. Percents of names of members of A.A.N.A. and A.S.A. included in questionnaires according to states.

purposes of the graphs listed as A.S.A.

3. Physicians (including "physician anesthetists" whose names were not in the A.S.A. Directory), for purposes of the graphs were listed as M.D.
4. Registered nurses (including R.N.A.'s whose names were not in the A.A.N.A. Directory), for purposes of the graphs were listed as R.N.
5. Others. The titles of this group in no way indicated any of the above categories and included:

"nurse" (as distinguished from R.N. on a few replies)

Doct. act. Osteopathy	displaced person
DMD	"office girl"
student nurses	"helper"
L. vocational nurse	"BS"
med. technicians	"special trained"
med. students	FACC
administrator	Ph.G. (sic)
L. pract. nurse	dental int.
pract. nurse	RN technician
nurses aid	dental student
dietitians	x-ray technician

Qualifications of Persons Administering Anesthesia

Group I: Active members of A.A.-N.A.

The Bylaws[5] of the American Association of Nurse Anesthetists list the following requirements for membership:

ARTICLE 1 MEMBERSHIP

Section 1. — Qualifications for Active Membership

Any person is eligible for membership as an Active Member who:

A. Is a graduate from an accredited high school or its equivalent,

B. Is currently validly registered as a professional, graduate nurse, and upon application for membership presents evidence of such current valid registration,

C. Is of good moral and ethical standing in the profession,

D. Presents evidence of:

1. Continuous experience in the administration of anesthetics since 1933 in approved hospitals; or

2. Clinical experience in the administration of anesthetics totalling at least five (5) years, which experience started before January 1, 1942, the adequacy of such clinical experience to be evaluated by the Credentials Committee and of a refresher course as specified by the Board of Trustees; or

3. Graduation from a school of anesthesia, which school,

   a. If graduation was prior to 1939, gave a course of at least four (4) months; or,

   b. If graduation was between 1939 and August 31, 1947, gave a course of not less than six (6) months and complied with the standards of this association; or,

   c. If graduation was after September 1, 1947, gave a course of not less than eight (8) months and complied with the standards of this association; or,

   d. If graduation was after January 1, 1951, gave a course of not less than one (1) year and complied with the standards of this association; and

E. Passes a qualifying examination as directed by the Board of Trustees of this association, unless waived by the Board.

(If date of graduation from any of the above courses was more than five (5) years prior to the date of application the candidate must present evidence of present clinical ability.)

Group II: Active members of A.S.A.

The Bylaws[6] of the American Society of Anesthesiologists gives the following definition in its categories of membership:

CHAPTER 1: MEMBERSHIP

"A physician who is engaged or specially interested in the practice of anesthesiology, who holds a degree of doctor of medicine or bachelor of medicine, who is licensed to practice medicine in the state in which he practices (excepting a doctor of medicine in the Armed Services of the United States of America or Canada

or in the Veterans Administration), and, who, if engaged in the practice of medicine, is an active member in good standing of his county medical society or its equivalent or, if not engaged in the practice of medicine, is of good ethical and professional reputation, shall be eligible to become an active member."

Since there is no way of checking the qualifications as anesthetists of any of the categories R.N., M.D. or others, no attempt has been made to do so. Those names were placed in these categories with every realization that some of these persons may be as well or better qualified as those whose names appear in the category of ASA and AANA, but it seems logical to assume that the larger percentage of persons who belong to their respective professional societies have been subjected to training that should qualify them above other persons who may not have had special training. For purposes of this survey the term "qualified" will designate categories I and II, those whose qualifications have been defined.

Using these categories, the Co-Directors of the Counselling and Testing Service, Roosevelt University of Chicago, Illinois, Alyce E. Graham and Richard E. Pasca, analyzed the data according to the following method:

"A description of the division of persons giving anesthetics into the 5 categories (AANA members, ASA members, R.N., M.D., etc.) was given above. We divided the hospitals into 4 categories as devised by the American Hospital Association, 0-49 beds, 50-99 beds, 100-249 beds, 250 beds and over. We tabulated the percent of anesthetics given by each professional category listed above and by various hospital size. A frequency distribution by professional category and hospital size was arranged. On the basis of this frequency distribution a median percent of anesthetics administered by each professional category by hospital size was obtained. Since all hospitals did not reply to this survey we projected these medians to determine the percent of coverage of all hospitals. We wanted to clarify the statistics as to the percent of qualified personnel administering anesthetics by various hospital size. We then computed the percent of coverage by AANA and ASA members for each size of hospital. The percent of coverage for the nation as a whole was obtained by a summation of the original tabulations and the computation of medians for each professional category."

Obstetric Coverage

The question concerning obstetric service was worded in a manner that made it difficult to analyze the returns. Since those questionnaires that did not have a reply to the question could not be categorized definitely as having obstetric service covered by the anesthesia department, or as having no anesthesia department, it was believed that conclusions would not be valid. From a casual survey, however, there are many departments of obstetrics that did not have anesthesia services by the members of the department of anesthesia, and in this group even a larger percentage of persons than were listed for surgical services were neither nurses nor doctors. To obtain accurate statistics we believe that a separate questionnaire should be sent to hospitals to obtain information concerning the anesthesia situation in the obstetric department.

Hospital Size in Relation to Anesthesia Personnel

From the data a chart (Figure 4) was constructed to show the percentage of hospitals that have even one member of the ASA or of the AANA or a combination of both. At least one member of ASA is available in only 10% of the 0-49 bed hospitals, 22% of the 50-99 bed hospitals, 53% of the 100 to 249 bed hospitals and 68% of the 250 bed and over hospitals.

At least one member of AANA is available in 29% of the 0-49 bed hospital, 59% of the 50-99 bed hospital, 78% of the 100-249 bed hospital and 74% of the 250 bed or over hospital.

The combined services of at least one ASA member or one AANA member or both shows 35% in the 0-49 bed hospital, 72% in the 50-99 bed hospital, 96% in the 100-249 bed hospital and 89% in the 250 bed or over hospital. The combination takes into account that in some instances several anesthetists work in more than one hospital and in many hospitals both ASA and AANA members contribute to the anesthesia service.

That the anesthesia service in each of these hospitals may not include full time coverage by qualified persons may be deducted from the fact that many anesthetists, both nurse and physician, are available for duty in more than one hospital.

As each name on the questionnaire was checked against the names in the directories of ASA and AANA, a name that appeared on more than one questionnaire was indicated, but such duplications were not added into

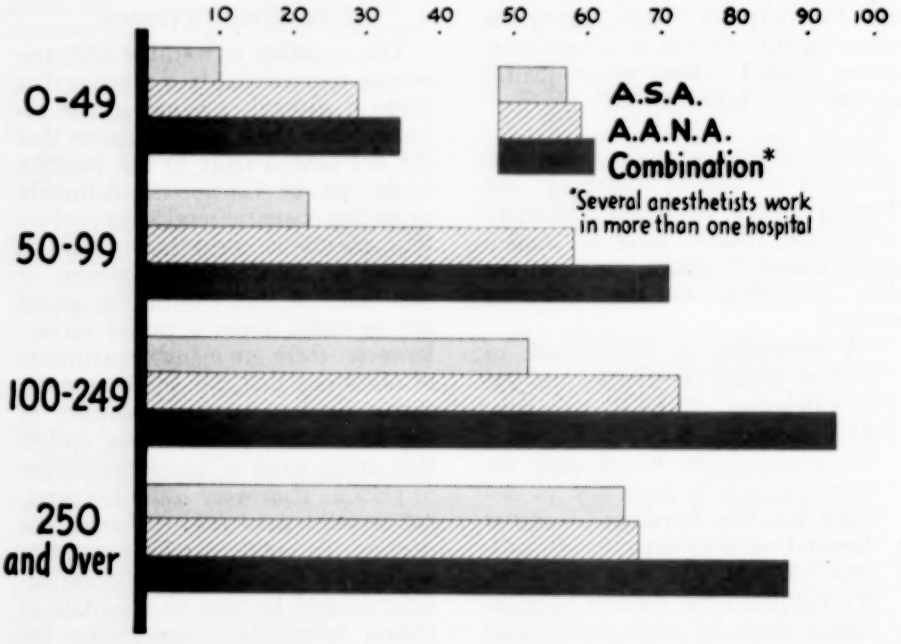

Figure 4. Percent of hospitals in each of four sizes that have anesthesia service available by even one member of ASA, AANA or a combination of both.

the total supplying the percentage of members involved in the sampling. The duplication of names may be of interest in that of the 3,250 names of AANA members, 139 appeared twice, 13 three times and 2 four times, and of the names of 1,638 ASA members 222 appeared twice, 62 three times and 13 four times.

Difference in Coverage by States

There is a wide range of anesthesia service by qualified persons, not only according to hospital size but according to state. (Figure 5)

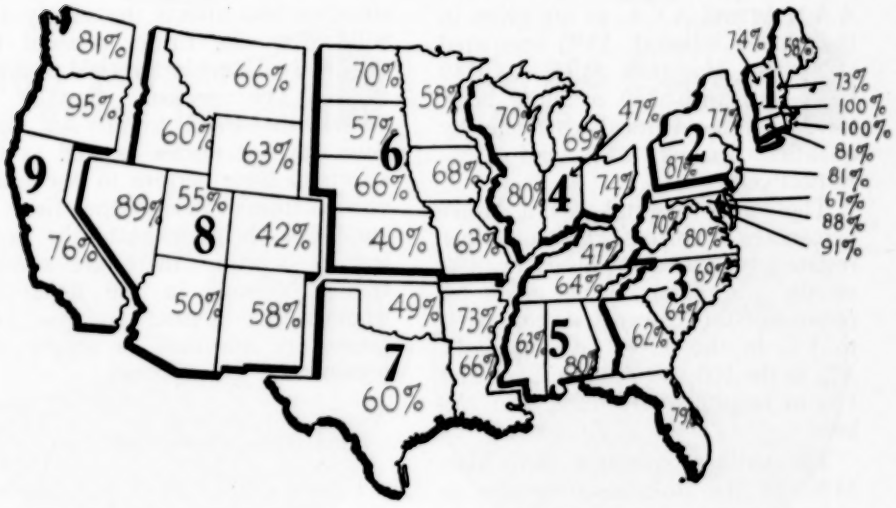

Figure 5. Percent of hospitals with at least one member of ASA or AANA available for anesthesia service.

Members of ASA or AANA are available in only 40% of hospitals in Kansas, the percent of persons in these categories ranging upward to 100% in Connecticut, Rhode Island and some United States territories and possessions.

Anesthesias Administered by Persons in Each Category

The data in the preceding figures would seem to indicate that there is progress being made in providing qualified persons for anesthesia service. Further study, however, reveals a somewhat less encouraging picture. The percents of the anesthesias administered by each person in each hospital were analyzed. In this part of the survey some of the data were not complete. Of 2,815 questionnaires used in the preceding analyses, 252 did not contain information usuable for this analysis. The remaining 2,563 questionnaires represent 35% of the entire number of hospitals contacted, and, therefore, seemed to constitute a valid sampling. The number of anesthesias administered

by each person was converted to percentage where percentages had not been given. The percentage of anesthesias given by each of the 5 categories of individuals was computed according to the method explained on page 229. The data are shown in Figure 6.

The small hospital, as is evident from the graphs, has far less than the national average of anesthesia service by qualified persons. Of the anesthesias administered in the hospital with 0-49 beds, only 29% are given by members of A.A.N.A. and A.S.A. In the hospital with 50-99 beds, almost twice as many anesthesias are given by members of A.A.N.A. and A.S.A. as are given in the smaller hospital: 53% compared to 29%. Hospitals with 100-249 beds, and with 250 or more beds, have almost identical coverage by qualified persons; 62% and 63%, respectively.

The small hospital has a larger percentage of persons who are neither registered nurses nor physicians, 4% of the anesthesias being given by persons of this category as compared to 1% in the 50-99 bed hospitals, 2% in the 100-249 bed hospitals, and 1% in hospitals with more than 250 beds.

The national averages show that 34% of the anesthesia service is given by members of the AANA, 18% by members of ASA, 19% by R.N.'s, 27% by M.D.'s, and 2% by persons who are neither registered nurses, nor doctors. Projecting the figures to national averages shows that 52% of anesthesias in the United States are administered by members of the AANA and ASA, 46% by physicians and nurses whose qualifications as anesthetists are unknown and 2% by persons who are neither nurses nor doctors.

With the continuing shortage of qualified anesthetists evidenced by the continuing demand, and with the increasing needs, it seemed well to know whether the demand was regional or general, and whether progress was being made in supplying the needs. That the need for anesthetists will increase seems to be a logical assumption in view of the number of new hospitals being built, the number of old hospitals being expanded to provide for the increasing hospital population; the increased number of births in hospitals; the number of aged patients; the increasing scope of prepaid hospital plans, and the broader scope of surgery. The nursing situation also affects the supply and will affect the future demand for qualified persons in the field of anesthesia. The present shortage of nurses, the trend toward an eight hour day for nurses as well as the efforts of some persons to discourage nurses from entering the field of anesthesia, help delineate the problem of whether there are enough trained persons in the field and whether the present training programs are adequate to supply the present and future needs.

This study shows that:

1. only slightly more than one-half of the anesthesias are administered by persons whose qualifications are known
2. the hospital with less than 100 beds does have fewer qualified persons to administer its anesthesia service than do larger hospitals, and
3. the combined services of members of ASA and AANA are not adequate to cover the demands in the hospitals of all sizes.

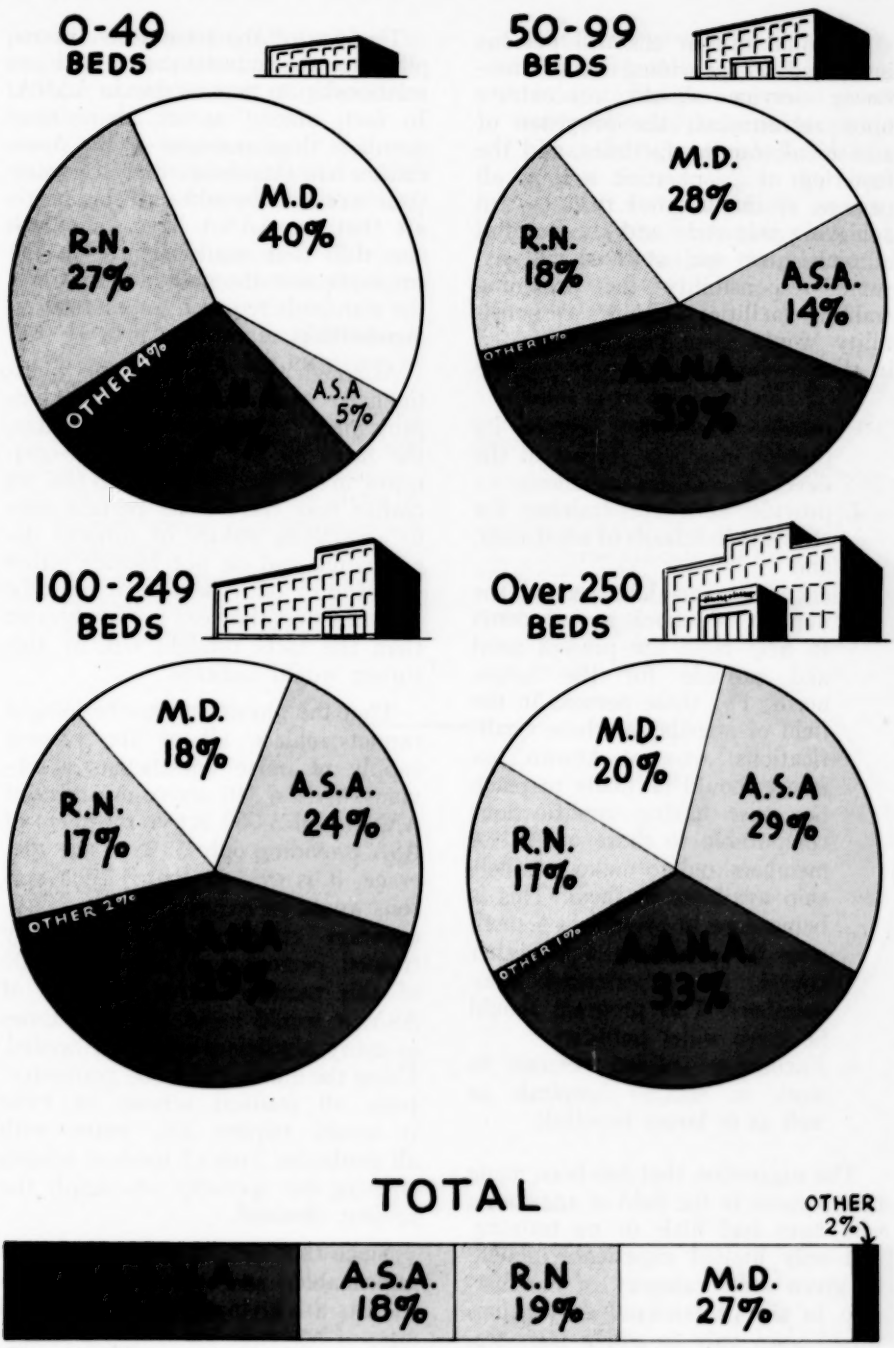

Figure 6. The percent of anesthesias administered by each of the five categories of persons in each of the four sizes of hospitals. The bar graph at the bottom of the page shows the national averages.

It would appear that all persons interested in providing good anesthesia service should concentrate upon recruitment; the provision of additional training facilities, and the fostering of cooperation among all persons in the hospital field toward achieving this end; and the hospital administrator and staff should assume responsibility for providing training facilities. AANA's responsibility would seem clearly indicated in that it should

1. provide means for keeping its present members abreast of the developments in anesthesia
2. provide adequate training for students in schools of anesthesia, and
3. encourage an increase in the number of schools and students to help meet the present need and provide for the future needs. For those persons in the field of anesthesia whose qualifications are not known, an effort should be made to reach the ones having qualifications comparable to those of AANA members and to make membership available to them. This is being done at present in a small way by providing a refresher course for experienced nonmembers. This program should be given wider publicity.
4. Encourage trained persons to work in smaller hospitals as well as in larger hospitals.

The suggestion that has been made that persons in the field of anesthesia who have had little or no training, and only limited experience, should be given some category of membership in the professional associations would seem only to add a false seal of approval.

The use of the term RNA in replies to the questionnaire had no relationship to membership in AANA. In fact, among nurses, more nonmembers than members of the Association had this designation following their names. It would seem appropriate that the AANA have some concise title that would signify to the employer that the individual has met the standards required for association membership.

Although the results of this questionnaire seem to indicate some improvement in anesthesia coverage, the facts do not warrant over-optimism in the past 6 years. When we realize that these facts pertain only to anesthesia service in surgical departments and do not include either obstetric or office service, it must be assumed that the need is even greater than the facts brought out by this survey would indicate.

That the physician anesthesiologist cannot replace all of the present supply of nurse anesthetists is obvious. With 6,340 active members of AANA and 3,000 active members of ASA providing only 52% of the coverage, it is evident that 17,963 persons would be required to give 100% coverage of anesthesia service by trained personnel. With only 3,000 of this number active members of ASA, it would mean almost 6 times as many physicians would be needed. Using the number of 6,800 graduates[7] from all medical schools in 1954 it would require 2½ years—with all graduates from all medical schools entering this specialty—to supply the present demand.

Since this turn of events is highly improbable, and since nurse anesthetists are giving good service in all sizes of hospitals for all types of surgery, it would seem logical for all

persons involved in this problem to cooperate in providing more well-trained nurses as well as physicians in this field.

Summary

A survey of the anesthesia service in United States hospitals has shown that:

1. 34% of anesthesias in all hospitals are given by members of AANA
2. 18% of anesthesias in all hospitals are given by members of ASA
3. 27% of anesthesias are given by physicians whose qualifications as specialists are unknown.
4. 19% of anesthesias are given by nurses whose qualifications are unknown
5. 2% of anesthesias are given by persons who are neither registered nurses nor physicians
6. Hospitals of less than 100 beds do not have anesthesia service by qualified persons in as great proportion as do larger hospitals
7. That the figures included in this report do not reflect the total picture since anesthesia service, other than for surgery, has not been covered by the survey.

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Modern Concepts of Balanced Anesthesia

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For almost 100 years after the introduction of anesthesia into medical practice, the various requirements of anesthesia in a given case were met by the administration of a single agent. This was made possible by the fulfillment, by ether and chloroform—the first two agents used extensively for surgical anesthesia—of the three most important requirements of anesthesia, namely, analgesia, sleep, and muscular relaxation. In the last few decades, however, it became evident that the use of a single agent for the production of these requirements is frequently accompanied by unnecessary depression of vital physiological mechanisms.[1]

In recent years there has been an increasing tendency to use a combination of several drugs for anesthesia, instead of a single agent. These are so selected that each drug employed is used for the purpose for which its pharmacological properties make it primarily suitable. For example, barbiturates are used to produce sleep, analgesics and nitrous oxide to produce analgesia and neuromuscular blocking agents to supply the necessary muscular relaxation.

The concepts of balanced anesthesia go back to Crile and Lower[2] and to Lundy.[3] A further impetus to the development of anesthesia was given by the introduction of cyclopropane and thiopental sodium. Since these agents did not always provide adequate muscular relaxation, neuromuscular blocking agents were introduced and are now being used extensively. A further development was the introduction of various narcotic analgesics for the supplementation of nitrous oxide-oxygen-thiopental anesthesia. This made possible the administration of smaller doses of barbiturates and resulted in earlier recovery of the patient. The most recent and logical step, leading to the perfection of balanced anesthesia, was the introduction of narcotic antagonists which, in appropriate dosage, counteract the respiratory depression produced by the narcotic analgesics.

To utilize fully the advantages of balanced anesthesia it is imperative that the agents used should not only be selective, but also short-acting and thus controllable. Consequently, whenever there is a choice, the

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shortest acting of the available drugs, suitable for the desired purpose, should be employed.

The technic of balanced anesthesia will depend on the nature of the case and the requirements that the anesthetist has to meet. In many cases it is only necessary to produce amnesia, sleep, and analgesia. In other cases good muscular relaxation should also be provided. Recently the maintenance of deliberately induced hypotension and hypothermia, as well as the sustenance of life in the presence of interrupted circulation to various vital organs, also became the province of the anesthesiologist.

In the present paper the technic of balanced anesthesia will be described both for cases in which muscular relaxation is not required and for cases in which it is needed. Since such requirements as pre-operative medication and induction of anesthesia are almost identical, irrespective of the need for muscular relaxation, these items will be discussed together.

Pre-operative Medication

Space does not permit detailed consideration of all aspects of pre-operative medication. A few important points however, might bear re-emphasis. Perhaps the most significant aspect of premedication is proper timing. All too often premedication is still ordered on call. This frequently results in too short an interval before induction of anesthesia, so that the premedication fails to exert the desired effect. Hence the patient is fully alert and apprehensive on arrival in the operating room. Pre-operative pain is present and the desired depression of the parasympathetic nervous system is not adequate. If, at this point, the anesthetist tries to correct the situation with an additional intravenous dose of a premedicating agent, then later in the course of anesthesia the original premedication, the corrective dose, and the anesthetic agent will act simultaneously and may cause undue depression of the patient's vital mechanisms.

Short-acting barbiturates should be given 90 minutes and analgesic and parasympatholytic agents 45 to 60 minutes before institution of anesthesia. To avoid the variability of absorption from the gastro-intestinal tract and the possibility of intracutaneous instead of subcutaneous injection, the drugs used for premedication are best given intramuscularly. When exact timing is not possible, premedication should be given too early rather than too late. If the effect of prematurely administered premedication has partly worn off, the situation can be remedied by giving an appropriate intravenous dose.

Besides the age, weight, and physical condition of the patient, his habits, such as alcoholism or barbiturate addiction and certain pathologic conditions such as hypothyroidism or liver or kidney disease, should also be taken into consideration. Alcoholics often become excited from short-acting barbiturates especially if they are in pain, and in such patients these drugs should be replaced by intravenous alcohol, or if this is not available, by paraldehyde or chloral-hydrate. Addicts need considerably larger doses of barbiturates and opiates for premedication. Patients with hypothyroidism or liver disease are very sensitive to short-acting barbiturates and opiates. In patients with liver disease short-acting barbiturates should be avoided and the dose of opiates must be decreased to 1/3 to 1/2 the normal dose. Long-acting

barbiturates, which are excreted through the kidneys, are not recommended in renal disease.

Preparation for Induction of Anesthesia

On the patient's arrival in the operating room an intravenous infusion is initiated with a No. 18 or 17 needle, using either a solution of 5% dextrose in H_2O or, if it is anticipated that the patient will require transfusion of blood during surgery, of 5% dextrose in 0.9% NaCl. From then on, all intravenous injections are administered through the rubber sleeve of the intravenous tubing.

Next, the patient's mouth and pharynx are sprayed with a suitable topical anesthetic agent (e.g. 1% tetracaine solution). Topical anesthetization of the mouth and pharynx and later that of the larynx is necessary because none of the agents used in balanced anesthesia are capable of depressing the irritability of the glossopharyngeal and the vagus nerves. Consequently, unless the upper air passages are topically anesthetized the patient will require much deeper planes of general anesthesia, to tolerate an oropharyngeal or nasopharyngeal airway or an endotracheal tube. Topical anesthetization will also help to prevent the development of laryngospasm and bronchospasm that occasionally complicate the induction of anesthesia with short-acting barbiturates.

The Maintenance of Analgesia and Sleep in Balanced Anesthesia

Two basically different methods can be used for the production of analgesia and sleep in balanced anesthesia. According to the first method,[4] thiopental sodium or a similar short-acting barbiturate is used for supplementation of nitrous oxide-oxygen anesthesia and a short-acting narcotic analgesic is administered only when the patient has already received a hypnotic dose of the barbituate. According to the second method,[5] relatively large doses of a short-acting narcotic analgesic are administered in conjunction with a narcotic antagonist. Subsequently, small doses of a short-acting barbiturate are used only if the analgesic and nitrous oxide-oxygen fail to produce the desired level of general anesthesia without undue depression of the patient's respiratory rate. The anesthetist must make up his mind before the induction of anesthesia whether he is going to rely primarily on a short-acting barbiturate supplemented with relatively small doses of a narcotic analgesic, or whether he will use larger doses of analgesic together with a narcotic antagonist and supplement these, as the need arises, with very small doses of a short-acting barbiturate. Indecision on the part of the anesthetist, resulting in the use of large doses of an analgesic after a hypnotic dose of a short-acting barbiturate, or in the use of large doses of a barbiturate after relatively large doses of an analgesic, will lead to undue depression of the patient. It has to be kept in mind that the narcotic antagonist is not capable of counteracting the respiratory depression caused by barbiturates. Furthermore, it also has to be remembered that patients are extremely sensitive to the respiratory effects of short-acting barbiturates after large doses of analgesics, even though a narcotic antagonist has been used. The situation is somewhat better when respiratory depression is caused by the administration of large doses of an analgesic given after large doses of a short-acting barbiturate. In these cases respiratory depression will be

effectively counteracted by subsequent administration of the narcotic antagonist.

Supplementation of Nitrous Oxide-Oxygen Anesthesia with Short-Acting Barbiturates and Small Doses of Analgesics

Anesthesia is induced by the injection of a 2½% thiopental sodium solution through the rubber sleeve of the intravenous tubing. The hypnotic dose of thiopental sodium should be administered slowly, in small increments, allowing at least 2 to 3 minutes for the injection of the total dose, which will vary from a few cc. to as much as 30 cc. The depth of anesthesia which should be tested frequently between each increment of thiopental sodium will be judged satisfactory when the patient tolerates the insertion of an oropharyngeal airway in the anesthetized pharynx.

Routine rapid administration of a single 10 or 20 cc. dose of 2½% thiopental sodium should be avoided. It must be realized that any drug, after intravenous administration, will temporarily accumulate in those organs which have the best blood supply, namely, the brain and the heart. Consequently, the rapid intravenous administration of thiopental might cause temporary accumulation in the central nervous system and the heart. This may result in temporary depression of the vital centers of the brain and interference with myocardial activity. In healthy individuals, especially if they are well oxygenated during induction, no lasting ill-effect will be caused by this temporary depression of vital functions. However, in patients with diseased myocardium or in whom the activity of the respiratory and circulatory center is already depressed, either by premedication or by pre-existing pathology, such methods of induction may easily cause prolonged circulatory and respiratory depression and occasionally may result in death. In debilitated patients most of the ill-effects of barbiturate anesthesia are caused by too rapid induction. To counteract the effects of possible respiratory and circulatory depression that might accompany the induction of thiopental sodium anesthesia, 100% oxygen should be administered during the induction period.

If the patient is breathing satisfactorily after the insertion of the oropharyngeal airway, the administration of nitrous oxide-oxygen in a 4 liter to 1 liter mixture can be initiated. If the patient's respirations are depressed after insertion of the airway, the administration of 100% oxygen should be continued until this difficulty is corrected. If the patient seems to resent the airway, a few additional cc. of thiopental sodium may be administered cautiously. The bag of the anesthesia machine is allowed to fill up 3 times in succession with the 4 liter to 1 liter nitrous oxide-oxygen mixture. In patients who might be sensitive to lack of oxygen or have a high oxygen consumption, a 3 liter to 1 liter or even a 2 liter to 1 liter nitrous oxide-oxygen mixture may be used for the induction of nitrous oxide-oxygen anesthesia. After the breathing bag of the anesthesia machine has been washed out at least 3 times with the selected gas mixture, the flow meters are set to deliver 500 cc. of nitrous oxide and 500 cc. of oxygen per minute. As previously described,[6] such a mixture will deliver 30± 5% oxygen and 70± 5% nitrous oxide to the average patient. Here again, if higher oxygen concentrations are desired or if the

patient's oxygen consumption is expected to be unusually high, 500 cc. nitrous oxide and 600 or 700 cc. of oxygen per minute may be administered. To obtain the maximum effect from the nitrous oxide-oxygen concentration used, it should be administered for at least 5 to 8 minutes before the beginning of surgery. Contrary to widespread belief, at least this length of time is necessary to obtain the maximum analgesic effect from a given nitrous oxide-oxygen concentration and thus to minimize the quantity of barbiturates and analgesics necessary for the supplementation of nitrous oxide-oxygen anesthesia.

If the patient's respiratory rate and depth are satisfactory after the administration of the hypnotic dose of thiopental sodium and saturation with the selected nitrous oxide-oxygen mixture, a small dose of a short-acting analgesic is administered intravenously before initiation of surgery. In our hands the shortest acting of all presently available potent analgesics, Nisentil® HCl proved to be most satisfactory.[4] Again, depending on the patient, the dose of Nisentil should be varied between 5 to 20 mg. If the depth of anesthesia is not satisfactory for the requirements of surgery a few minutes after the administration of the initial Nisentil dose and the patient's respiratory rate is above 12 per minute, another small dose of Nisentil may be administered. The Nisentil administration can be continued until either satisfactory depth of anesthesia is obtained or until the patient's respiratory rate decreases to 12 or below. If satisfactory operating conditions cannot be obtained without the depression of the respiratory rate below 12, this is a sign that the patient needs more thiopental sodium. In such cases thiopental sodium is administered cautiously in 1 to 2 cc. increments until the desired depth of anesthesia is attained. If the respiratory rate increases above 12, it should be endeavored to maintain the necessary depth of anesthesia by cautious administration of small doses of Nisentil. It will soon become evident how large a dose of Nisentil is tolerated without undue respiratory depression. This dose should then be administered at the necessary intervals which may vary from 6 to 20 minutes. Usually, as the anesthesia progresses, the intervals between subsequent fractional doses of Nisentil may be increased. During very long operations it will be necessary occasionally to administer an additional 1 to 2 cc. of thiopental sodium. In general, no more Nisentil has to be given after the start of the wound closure.

Since intravenously administered Nisentil has a relatively short-lasting effect and since most of its action is dissipated within 20 minutes after intravenous administration, most patients will regain consciousness within a short period after discontinuation of nitrous oxide-oxygen.

With the technic described, which was used in a large series of patients, 60-90% reacted to stimulation at the termination of surgery.[4] In general, the longer the duration of anesthesia, the higher the percentage of patients who react to stimulation. This seemingly paradoxical finding is due to the fact that the longer the anesthesia, the more time is available for redistribution and detoxification of the initial dose of thiopental sodium and this results in a more speedy recovery of consciousness.

It must be emphasized that both thiopental sodium and Nisentil used for supplementation of nitrous oxide-

oxygen anesthesia will always produce some depression of the respiratory rate and depth. Consequently it is essential that the anesthetist assist the patient's spontaneous respiratory effort throughout anesthesia. This can best be accomplished by supplementing the patient's tidal volume to 500 to 600 cc. by gentle pressure exerted on the breathing bag of the anesthesia machine during the inspiratory period. With a tidal volume of 500 to 600 cc. and a respiratory rate above ten, the minute volume and what is more important, the alveolar ventilation rate, will be adequate to prevent accumulation of CO_2. Too vigorously assisted respiration or too vigorously controlled respiration of the apnoeic patient might lead to hypocapnia.

If, because of the contour of the patient's face or for some other reason, a tight fit in the respiratory circuit cannot be obtained, it might be necessary to use higher flows of nitrous oxide and oxygen to maintain an adequate gas volume in the breathing bag of the anesthesia machine. In that case, the flow of nitrous oxide and oxygen must be so adjusted that the resulting mixture will supply approximately 30% oxygen and 70% nitrous oxide. The flow meter settings necessary to give such a gas mixture at different rates of total flow are shown in table 1. The principles of nitrous oxide-oxygen administration in closed circuit were described in greater detail in a previously published paper.[6]

If the closure of the anesthetic circuit is good, then the administration of 500 cc. of nitrous oxide and 500 cc. of oxygen per minute will lead to the accumulation of gas and the distention of the breathing bag of the anesthesia machine. This will hinder the patient's expiratory efforts; consequently, whenever the breathing bag is more than 3/4 full, the bag should be partially emptied through the exhaust valve of the anesthesia machine.

TABLE 1

FLOW METER SETTINGS NECESSARY TO OBTAIN APPROXIMATELY 70% N_2O-30% O_2 MIXTURES IN THE INHALED GAS MIXTURES WITH VARIOUS TOTAL FLOW RATES

TOTAL FLOW cc. PER MIN.	FLOW METER SETTING cc. PER MIN. N_2O	O_2
1000	500	500
1500	850	650
2000	1200	800
3000	1900	1100
4000	2600	1400

Supplementation of Nitrous Oxide-Oxygen Anesthesia with Analgesics, Narcotic Antagonists, and Small Doses of Short-Acting Barbiturates.

The drugs used with this technic for supplementation of nitrous oxide-oxygen anesthesia are levallorphan

tartrate, a narcotic antagonist (structurally related to levo-Dromoran® (see figure 1) and Nisentil. As soon as the intravenous infusion is

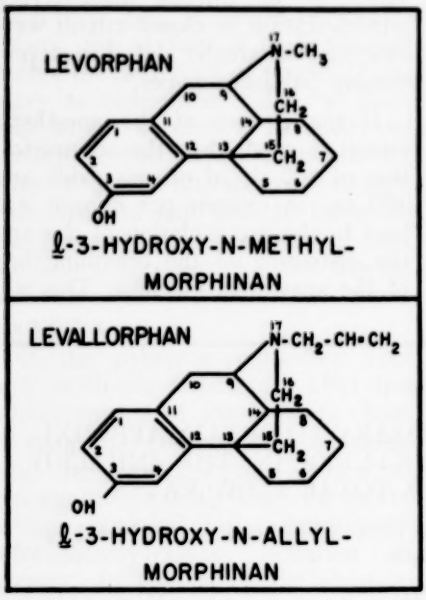

Figure 1

The Structural Formulae of Levo-Dromoran® (Levorphan) and Levallorphan.

started, levallorphan (0.01 mg./lb. of body weight) is administered intravenously. At least 6 to 8 minutes should be allowed between the initial doses of levallorphan and Nisentil. The initial dose of Nisentil is about 0.4 mg./lb. of body weight. Independent of the patient's weight, the maximum initial dose of levallorphan should be limited to 1.5 mg. and that of Nisentil to 80 mg. Nisentil should be injected slowly; at least ½ to 1 minute should be taken for the injection of the total initial dose. In old and debilitated patients the initial dose of Nisentil should be decreased to 2/3 or 1/2 but the initial dose of levallorphan should be the same as in younger and stronger individuals. Following the injection of the initial Nisentil dose, the administration of nitrous oxide-oxygen is started in the manner described above. After the nitrous oxide-oxygen mixture has been given for a few minutes, an oropharyngeal airway is inserted in the anesthetized pharynx. Patients, although awake and obeying commands, will readily tolerate the airway at this time, in contrast to patients in whom thiopental sodium was used as supplementing agent. In the exceptional patient who does not tolerate the airway, 2½% thiopental sodium should be administered in 1 to 2 cc. increments, 1 to 2 minutes apart, until the oropharyngeal airway can be inserted. When this is accomplished, the breathing bag of the anesthesia machine is washed out again 3 times with the selected nitrous oxide-oxygen mixture and then the flow rate of both the nitrous oxide and oxygen is decreased to 500 cc. per minute.

The patient's sensitivity to pain should be tested before the beginning of surgery. If the patient reacts to painful stimuli, but the respiratory rate is above 16, an additional 5 to 20 mg. dose of Nisentil may be administered. If suitable depth of anesthesia cannot be obtained by additional doses of Nisentil without depressing the respiratory rate, this is an indication that the patient needs small doses of thiopental sodium, which should be administered in 1 to 2 cc. increments, 1 to 2 minutes apart. The effect that can be obtained from a very small dose of thiopental sodium is indeed surprising. In most instances the administration

of 2 to 4 cc. of 2½% thiopental sodium will produce adequate depth of general anesthesia at this time. The administration of larger doses of thiopental sodium or of as little as 4 cc. in one single dose will very frequently produce respiratory depression. As mentioned, respiratory depression of this type is not amenable to treatment with levallorphan, and if it occurs respirations should be controlled until the patient resumes spontaneous respiratory function.

Anesthesia can be maintained at a satisfactory level by the administration of fractional 5-20 mg. doses of Nisentil, administered, 8 to 20 minutes apart, as required. Additional 1 to 2 cc. doses of thiopental sodium will have to be administered only if adequate depth of anesthesia cannot be maintained without depression of the respiratory rate below 12. The anesthetist will be gratified to see that even in the presence of markedly decreased respiratory rate, the respiratory depth will usually be adequate. Nevertheless, as with the previously described method, assistance of the patient's spontaneous respiration is essential with this technic.

In contrast to Nisentil, levallorphan is a relatively long-acting drug. Consequently, the administration of additional doses of levallorphan are usually unnecessary unless the duration of anesthesia exceeds 2 to 3 hours. For longer procedures it is frequently necessary to administer additional 0.5 to 1 mg. doses of levallorphan.

As with the alternate method described, it is usually unnecessary to administer additional Nisentil after beginning of the wound closure. Consequently, the patient will usually regain consciousness very rapidly after discontinuation of nitrous oxide-oxygen. In a large series of cases recently reported,[6] the number of patients who reacted to stimulation at termination of anesthesia was well over 90%. Not only do the patients react to stimulation postoperatively, but most of them show a surprisingly high degree of alertness immediately after cessation of nitrous oxide-oxygen anesthesia. They are conscious of their surroundings, are able to answer questions, and very frequently inquire about the outcome of the operation.

The Use of Muscle Relaxants in Balanced Anesthesia.

The technic of administering a muscle relaxant will depend on the drug selected and on whether the muscle relaxant is necessary only for facilitation of endotracheal intubation or for maintenance of prolonged muscular relaxation during surgery. In order to evaluate the patient's requirements of the various drugs used for maintenance, it is essential that no muscle relaxant be administered until, after the induction of anesthesia, the anesthetist satisfies himself that the patient is breathing adequately, that no centrally induced respiratory depression is present, and that the level of general anesthesia is not too light, which would lead to breathholding of the patient.

The Use of Muscle Relaxants for Endotracheal Intubation.

When muscle relaxants are to be employed for the sole purpose of facilitating endotracheal intubation, it is logical that the shortest acting muscle relaxant, succinylcholine (Anectine), should be used.[7] In gen-

eral, the slow (30 seconds) intravenous administration of Anectine (0.2 to 0.3 mg./lb. of body weight) will produce good relaxation and apnea, in about 30 seconds after the end of the injection. This effect will last for an average of 2 to 4 minutes. Patients should be hyperventilated with 100% oxygen during and after the injection of succinylcholine. If the succinylcholine is injected over a period of 30 seconds, muscular fasciculations will be mild or absent in the majority of patients. Hyperventilating the patient with 100% oxygen will fill the respiratory tract with oxygen and by diffusion produce sufficient oxygenation during endotracheal intubation, even if this would take as long as 1 minute. If the endotracheal tube cannot be introduced within this period, the patient should be hyperventilated again with 100% oxygen for ½ minute and endotracheal intubation again attempted. If by this time the effect of the initial dose of succinylcholine has worn off, ½ or 2/3 of the initial dose will reestablish muscular relaxation to accomplish endotracheal intubation. After visualization of the larynx and before insertion of the endotracheal tube, the cords and the trachea should be sprayed with a suitable topical anesthetic agent. Following the insertion of the endotracheal tube the patient is again hyperventilated with 100% oxygen for a few seconds and then nitrous oxide-oxygen is administered as described above, either through a face mask placed over the endotracheal tube or through connections directly attached to it. In either case, care should be taken not to move the endotracheal tube unnecessarily because the stimulus of a moving tube will frequently cause reflex breathholding in the lightly anesthetized patient. As long as apnea persists the patient's respirations should be controlled by administering 500 to 600 cc. of the selected gas mixture by squeezing 10 to 12 times per minute on the breathing bag of the anesthesia machine. From then on, assisted respiration should be used. Too vigorous controlled respiration during the period of apnea might produce hypocapnia resulting in prolonged apnea which will make the resumption of the patient's spontaneous respiratory efforts difficult.

In patients in whom undue sensitivity to succinylcholine can be expected (patients with liver disease, malnutrition, severe anemia, cachexia due to malignancy, etc.) the dose of succinylcholine has to be reduced markedly. It is a safe practice to administer an initial dose of 5 mg. of succinylcholine to these patients. Frequently, this test dose proves satisfactory for the facilitation of endotracheal intubation. However, if it does not, then judging from the effect of the initial succinylcholine dose, additional doses may be given until conditions suitable for endotracheal intubation are attained. In our experience, alarmingly prolonged apnea will not follow the administration of the usual doses of succinylcholine in patients with increased sensitivity to this agent. The apnea may then last as long as 8 to 15 minutes, but it will wear off without any ill effect if the patient's respiratory exchange is maintained adequately during the apnoeic period.

When succinylcholine is not available, one of the long-acting muscle relaxants may be used to produce muscular relaxation for endotracheal intubation. In general, the initial dose of a long-acting relaxant is proportional to the body weight; it is greater in muscular individuals

TABLE 2

THE AVERAGE INITIAL AND FRACTIONAL DOSES OF LONG-ACTING RELAXANTS

	INITIAL DOSE (MG.)	FRACTIONAL DOSE (MG.)
d-TUBOCURARINE	12.0 to 15.0	4.0 to 6.0
DIMETHYLTUBOCURARINE	4.0 to 8.0	2.0 to 3.0
MYTOLON	12.0 to 15.0	4.0 to 6.0
FLAXEDIL	80.0 to 100.0	30.0 to 40.0
SYNCURINE	2.5 to 4.0	1.0 to 1.5

than in asthenics, in males than in females, and in the young and middle-aged than in the aged. Of the commonly encountered pathological conditions dehydration and electrolyte imbalance, cachexia due to malnutrition, chronic infection or neoplastic disease increase sensitivity to the long-acting relaxants and necessitate significant decreases of dosage.[1] The recommended initial and fractional doses (see below) of these relaxants are summarized in table 2.

Since the size of the initial dose of the relaxant is influenced by numerous factors and is selected primarily on an empirical basis, the personal experience of the anesthetist is of greatest importance, but error in judgment cannot be avoided even by the most experienced. If the estimated dose is too high, apnea lasting anywhere from 10 to 30 minutes or more may result. If, on the other hand, the dose was underestimated, smooth endotracheal intubation will not be accomplished. In such instances the injection of a fractional dose of the relaxant will be required. It is much safer to inject a relatively smaller dose followed by another fractional dose if necessary, than to risk prolonged apnea caused by an overdose.

THE USE OF MUSCLE RELAXANTS FOR THE MAINTENANCE OF PROLONGED RELAXATION.

The technic of maintenance of prolonged muscular relaxation during surgery will be considerably different if long-acting muscle relaxants are used than if succinycholine is administered by continuous intravenous drip. In general the patient's response to the initial dose of a long-acting relaxant is a good guide to the size of the first fractional dose and to the time of its administration. Only after the response to both the initial and the first fractional dose has been observed, will the effect of subsequent fractional doses become more or less predictable.

In general, diminishing muscular relaxation of the operative field, which is usually accompanied by an increase in the depth of respiration, indicates the necessity for administration of a fractional dose. To insure optimal operating conditions, it is a good practice to administer a dose of relaxant just before the beginning of the closure of the peritoneum. The size of this dose will depend on the size of the fractional doses, the interval between the administration of these doses, and finally on the time

since the administration of the last fractional dose. For example, if the fractional doses of gallamine were 30 mg., administered 20 minutes apart, and if the last dose was given 10 minutes before the beginning of the peritoneal closure, the dose to be administered at this time should be 10 mg. Usually, no more muscle relaxant will be required after the dose prior to the peritoneal closure.

When muscle relaxants are administered correctly, respiratory depression will seldom be encountered at termination of surgery. If, despite all precautions, respiration is inadequate at termination of surgery, it is by far the safest to oxygenate the patient by assisted respiration until satisfactory spontaneous respiration is re-established. Should it become necessary for some reason to administer an antagonist, the following precautions are recommended: The initial intravenous dose of neostigmine depending on the size and physical condition of the patient should be between 0.5 to 1.5 mg. in adults and proportionately less in children. Atropine sulfate 0.4 to 0.6 mg. should be mixed with the neostigmine. The injection should be given very slowly. If, after 5 minutes, the effect of this dose is not satisfactory, one half of the initial dose may be administered. The initial intravenous dose of Tensilon® is 5 to 10 mg. An 0.3 to 0.6 mg. dose of atropine sulfate, although not absolutely essential, is recommended to decrease the possibility of the occurrence of side effects of this drug. If the desired effect is not obtained within 2 minutes, additional 5 mg. doses of Tensilon may be given. Patients to whom antagonists to muscle relaxants have been administered, should be kept under close supervision for 30 to 60 minutes after return of adequate respiration, to ensure against the possibility of recurarization.

It has to be remembered that both neostigmine and Tensilon, with the exception of some very unusual cases, are only effective against the non-depolarizing muscle relaxants like d-tubocurarine, dimethyl tubocurarine, and Flaxedil.® Both neostigmine and Tensilon are less effective against Mytolon,® not at all effective against decamethonium and will prolong the duration of action of succinylcholine and similar compounds.

No commercially available antagonists to decamethonium or succinylcholine are at the disposal of the anesthetist.

When succinylcholine is to be employed for the production of prolonged muscular relaxation, it should be administered by continuous intravenous drip, in an 0.1 to 0.2% solution.[8] Following induction of anesthesia as described above, oxygen or nitrous oxide-oxygen is administered through a face mask and a succinylcholine drip is instituted at a fairly rapid rate, (180 to 240 drops of an 0.1 to 0.2% solution which corresponds to about 9 to 24 mg. per minute) in patients with normal plasma cholinesterase activity. This is given at a considerably slower rate of the more dilute solution in patients with expected low plasma cholinesterase activity. Muscular relaxation with depression of the respiratory tidal volume usually develops in 2 to 3 minutes. The development of the effect of succinylcholine can be followed by palpation of the breathing bag. When the decrease of the respiratory depth indicates adequate relaxation, the drip is either slowed to the expected maintenance rate (40 to 100 drops per minute), or stopped in sensitive patients and endotracheal

intubation is performed. Should apnea develop during or after intubation, the drip is discontinued and controlled respiration is maintained by administering 500 to 600 ml. of gas mixture by intermittent positive pressure, 12 to 16 times per minute, until spontaneous respiration returns. The drip is re-instituted at this time at a rate that is expected to give adequate relaxation without apnea. This rate will vary between 20 to 120 drops per minute depending on the size, age, physical condition, and plasma cholinesterase activity of the patient.

If the selected drip rate produces excessive depression of the tidal volume or apnea, the drip is immediately discontinued until the return of adequate respiration when it is re-instituted at a slower rate. Conversely, if the relaxation is inadequate, the drip is speeded up until adequate relaxation is obtained at which time it is adjusted to a somewhat faster rate. As is evident it is essential for maintenance of adequate relaxation with apnea, to know at all times the drip rate of the succinylcholine solution and to have a clamp which allows the easy and gradual control of this drip. Both requirements can be met most easily if the anesthetist has at his disposal a stop watch for rapid estimation and a tunnell clamp* for the rapid regulation of the drip rate. Counting 5 drops, if the rate is slower than 90 drops per minute, or 10 drops at a higher rate, allows the estimation of the drip rate in less than 10 seconds. If a tunnell clamp is not available, the use of 2 ordinary screw clamps can be substituted. The drip rate can be adjusted coarsely with the first one, and fine adjustment can be made with the second.

With this method of succinylcholine administration muscular relaxation can be regulated to the needs of surgery within 30 to 60 seconds and normal respiratory exchange can be established within 2 to 3 minutes after discontinuation of the drip. Satisfactory muscular relaxation can be obtained without apnea in patients with normal, or decreased, or without plasma cholinesterase activity. The only difference between patients with normal and low cholinesterase activity will be that a much slower drip rate will be necessary in the latter group and that the return of normal respiratory exchange might be prolonged by a few minutes.

Whenever controlled respiration is used with succinylcholine because of the requirements of surgery or the desire of the surgeon, care must be taken that the plasma level of succinylcholine should be just adequate to produce apnea. This can be achieved by discontinuing the drip at frequent intervals and observing the time necessary for the return of the first effort of spontaneous respiration. This should occur within 1 to 2 minutes, if no hypocapnia is produced by excessive controlled respiration. When the time necessary for spontaneous respiration is greater than 1 to 2 minutes the drip rate should be decreased accordingly. For smooth functioning of this method of "back titration" the recommended minute volume of respiratory exchange maintained by controlled respiration should be between 6 to 8 liters with a respiratory rate of 12 to 16 per minute.

With the recommended technic good muscular relaxation can be obtained without the development of prolonged postoperative apnea. If,

*Tunnell clamp can be obtained from Harvard Instrument Company, Dover, Massachusetts.

however, unnecessarily large doses of succinylcholine are administered over a prolonged period, then prolonged postoperative apnea will develop in a high percentage of cases. This prolonged apnea is caused by the accumulation of non-hydrolyzed succinylcholine in the plasma and also by its primary breakdown product, succinylmonocholine. One has to keep in mind that the prevention of apnea during anesthesia is the only sure way to avoid prolonged postoperative apnea. If apnea develops during the administration of succinylcholine it should be discontinued immediately until the return of spontaneous respiration of the patient.

Summary and Conclusions

The use of balanced anesthesia necessitates the simultaneous administration of several drugs. Thorough knowledge of the pharmacological effects of these drugs and rigid adherence to the principles of their safe administration are essential in the application of the methods described. When used improperly instead of balanced anesthesia, "shot-gun" anesthesia will result, which might endanger the patient's life. When correctly used, balanced anesthesia will cause minimal interference with the patient's vital mechanisms during anesthesia and will result not only in a rapid postoperative recovery of consciousness, but also in an uneventful postoperative course.

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Fatal Circumstances in 384 Anesthesia Deaths

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A study of deaths partially or wholly attributable to anesthesia is of interest not out of morbid curiosity, or to fix or absolve individuals from blame, but to guide teaching and investigation. We should teach those principles and methods least likely to be fatal yet most likely to produce optimum operating conditions. Research should be directed toward discovering those agents and technics most likely to be similarly effective.

Data herein presented derives from that assembled for "A Study of Deaths Associated with Anesthesia and Surgery", based on a study of 599,548 anesthesias in ten institutions from 1948 to 1952, inclusive. Thirty surgeons and anesthetists recorded all anesthesias given in their hospitals during these five years and studied and classified every death on their surgical services at the time it occurred. Those fatal cases in which anesthesia was considered to play a primary or contributory role were completely summarized and sent to the central office together with the basic numerical information. Thus we were able to establish more accurately than heretofore an anesthetic death rate. The basic data and statistical aspects of this study have appeared in the July 1954 Annals of Surgery.[1]

Of special interest to this group may be a description of those clinical anesthetic circumstances which were considered important by these thirty individuals in the fatal outcome of these surgical patients.

A definition of "Anesthesia Death" is difficult to state simply. However, if we are to advance in our learning, if we are to improve our record, we must study those circumstances which may only possibly have contributed to death. As this description advances it will become clear what elements may combine to produce an "Anesthesia Death".

A word of caution in drawing conclusions is appropriate from the start. Although one does not deliberately

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set out on a frequently fatal course of procedure it should not be implied that every fatal circumstance which we are about to describe should be avoided at all costs. So that the surgeon may accomplish a curative operation it may be necessary to undertake an anesthetic procedure of known hazard—the so-called "calculated risk". But it is of prime importance that we know, both the anesthetist and the surgeon, what anesthetic procedures are likely to be fatal, so that we may balance the added anesthetic hazard against the added advantage to the surgical cure.

The Data

You may be eager to hear a clinical summary of each of these 384 "Anesthesia Deaths". Even if each case were boiled down to two or three lines it would require the recitation of 47 closely typewritten pages and leave you with nothing but a jumble of anesthetists' nightmares. I have therefore attempted to categorize common fatal situations under three principle headings which concern us most in teaching and practice: neglect of **basic principles**; errors in **anesthetic management**; and errors in **recovery care**.

Although, of course, the whole story is not told by a single fatal factor, I have tried to assign the single most important factor in each case and list its occurrence under the appropriate heading.

Perusal of the three tables on the following page will indicate wherein anesthesia lay at fault.

Discussion of Data as a Whole

It is worthwhile to make some points here that will not be brought out in the summary.

Although anesthesia may seem cut and dried to the uninformed surgeon, he must realize that anesthetists make allowance for certain diseases. Errors in surgical diagnosis and especially errors of omission in diagnosis of non-surgical disease leave the anesthetist unprepared to combat them and unnecessarily add to the anesthetic hazard.

There are still sporadic attempts to operate under heavy sedation, but this medieval custom is being rather widely replaced by anesthesia. But the relaxant drugs are tempting some anesthetists to give up using anesthetics. It is well to realize this practice can not only lead to embarrassing prolonged apnea, but to death.

The importance of toxic action on the liver of some of the commonly used anesthetics is gradually going out of fashion and being replaced by some better founded factors.

Premedication is often a good test of the patient's tolerance for anesthesia and operation. It is well to heed the warning of excessive depression. The better part of valor is to return the patient to the ward, study his coronary circulation and blood volume and return him at a later date under less medication.

A partial explanation of the shockingly high anesthetic death rate among infants and children uncovered by the parent study of this paper may lie in Table II. Fully one third of the misapplications of anesthetic apparatus, mismanagement of depth of anesthesia and fluid administration was in infants and children. Here is a focus for attention.

Not infrequently so multiple were the anesthetic errors that no one could be singled out; so arose the heading "Compounded errors." Those of us charged with the responsibility of teaching should hear this: if an anesthetist is in trouble he is likely to be in more than one trouble and needs help from the start.

Table I
BASIC PRINCIPLES

	Examples
A. Preparation of patient	
1. Error in surgical diagnosis leading to mismanagement of anesthesia (pheochromocytomas - 2)	6
2. Failure to diagnose recent coronary occlusion	4
3. Failure to empty stomach	20
4. Failure to combat anemia, hypovolemia or dehydration	4
B. Choice of anesthesia	
1. Spinal anesthesia for senile, hypertensive, hypovolemic, anemic patient	19
2. Respiratory depressant anesthesia for patients with impaired respiration: obstructed airway or pulmonary disease	18
3. Cardiac depressant anesthesia (pentothal, cyclopropane) in patients with cardiac disease or cardio-vascular depression	16
4. Use of agents toxic to diseased liver	7
5. Failure to use sedatives for sedation, analgesics for anesthesia and relaxants for relaxation	6

Table II
MANAGEMENT OF ANESTHESIA

	Examples
1. Poor judgment in starting anesthesia and operation in patients unduly depressed by premedication	8
2. Chronic hypoxia leading to cardiac arrest and/or cerebral death (too high concentration nitrous oxide; failure to use oxygen; unexplained cyanosis; postoperative cerebral death; sudden, unexplained cardiac arrest; chronic hypotension)	29
3. Inability of anesthetist to compensate for inadequate respiratory exchange (depression or obstruction)	40
4. Use of respiratory depressants to compensate for inability of anesthetist to compensate for impaired respiration	4
5. Failure to use anesthetic apparatus (machine or endotracheal tube) suitable to patient and operation (infants - 3; children - 3; adults - 4)	10
6. Failure to recognize signs of anesthesia, or overdosage (infants - 15; children - 12; adults - 60)	87
7. Blood and fluid mismanagement: under or overtransfusion, over hydration. (Infants and children - 6; adults - 16)	22
8. Too much anesthesia and operation	16
9. Technical mistakes	
a. Intravenous air from pressure transfusion	4
b. Use of blood intended for another	3
c. Mixed tanks	3
d. Regional anesthetic accidents	5
e. Excessive positive pressure	3
10. Compounded errors (infant and children - 3; adults - 9)	12

Table III
RECOVERY CARE

	Examples
1. Respiratory depression or obstruction	20
2. Vomiting and aspiration	3
3. Premature moving of patient	6
4. Failure to treat shock or hemorrhage	4
5. Failure to diagnose post-anesthetic or post-operative complications	4
6. Wrong identification of oral or nasal tubes	1

In Table III, although recovery rooms have done much to avoid accidents, they are still occurring, sometimes in the recovery room but more often after hours, when an operation lasts into the evening or on holidays. Special vigilance is needed here on the part of the anesthetist to be sure adequate care is provided.

Summary of Data

To help crystallize this polyglot of anesthetic mishaps, Tables IV and V present a classification into those that should prove useful in teaching and research.

Table IV lists the important causes of "anesthetic death" that we should have been able to avoid: approximately 30% of the total deaths. Although in each individual case the patient was fatal. Another approach to surgical cure might have been more successful.

The hazard of a full stomach is constantly emphasized yet still overlooked in 5% of the deaths. The discomfort of gastric lavage is a small price to pay for a patient's life.

A basic premise in all anesthetic teaching is the folly of choosing spinal anesthesia, respiratory depressants and cardiac depressants for those patients whose diseases make them unusually susceptible. Yet in 15% of the deaths an attempt was made to "get away with it". Teachers should take heed that their precepts are indeed life saving.

5% of technical mistakes may be the irreduceable minimum of human error: yet when error is fatal it is hardly acceptable.

Table IV
OUTSTANDING AVOIDABLE CAUSES OF DEATH

	Cases	% of 384 deaths
Failure to empty stomach	20	5%
Spinal anesthesia for the senile, hypertensive, hypovolemic or anemic patient	19	5%
Respiratory depressant anesthesia for patients with respiratory impairment	18	5%
Cardiac depressant anesthesia for patients with cardiac impairment	16	4%
Technical mistakes	18	5%
Respiratory failure during recovery	20	5%

Table V
OUTSTANDING CURABLE CAUSES OF DEATH

	Cases	% of 384 deaths
Overdosage or excessive depth of anesthesia	87	23%
Inability to compensate for inadequate respiration	40	10%
Chronic hypoxia	29	8%
Blood and fluid mismanagement	22	6%
Excessive anesthesia and operation	16	4%

circumstances may have warranted undertaking these procedures of known hazard, the outcome for each

During recovery care an improved guard is needed against respiratory failure due most frequently to airway

obstruction and post-anesthetic respiratory depression.

Table V displays those causes of "anesthetic death" that are not so easily recognized nor so easy to avoid, yet account for 50% of our mishaps.

Improved training can certainly help to reduce miscalculation of anesthetic dosage or misinterpretation of the signs of anesthesia. It should also improve the ability of the anesthetist to maintain respiratory exchange.

However the last three categories and probably a large part of the first require some better means than the routine ones of observing the state of the patient. Cyanosis is a poor guide to hypoxia; blood pressure and pulse (often neglected in infants and children for want of suitable apparatus!) does not indicate well cardiovascular function; time is an inadequate measure of severity of operation.

We think nothing of spending for airplane equipment $500 for an altimeter or $2,000 for radar equipment to protect healthy people from traumatic shock, yet we are most reluctant to spend even $300 to assure a sick patient of an adequate respiratory exchange. If this tidal exchange meter only lasted one year and if it were only used on 300 patients, it would cost each patient one dollar to be sure he breathed enough under anesthesia and not only lived, but could do cross-word puzzles when he awoke! This is undoubtedly a homespun simplification, but if almost 50% of our deaths are to be avoided we must have the technical armamentarium with which to do it. Both research into a means and education to its use are vitally needed.

Summary and Conclusions

What has this unprecedented study produced?—something new—a little; something old—a lot.

In a sense you may have been disappointed to hear restated again and again principles you have already been taught. In a sense this is discouraging: we never seem to learn. In a sense it is encouraging: the principles we were taught are still of paramount importance—we must not lose sight of them in the momentary excitement of new drugs and methods.

What is new? Never has the hazard of infant anesthesia been brought out so strongly. We must not neglect anesthetic care of infants because they are small and difficult to adapt to adult methods. They need blood pressure, pulse and respiration recordings, they need blood replacement, they need adequate means of respiratory exchange: in fact they need it more than adults because small changes are so important to them.

Research must help to spare us from almost 50% of anesthetic deaths: investigation to establish prefatal signs of hypoxia, of depth of anesthesia, of fluid derangement; inventive genius to produce simple, inexpensive apparatus for their measurement; and an enlightened profession to accept the new ideas.

A sense of good judgment is needed—and as the specialty ages it will mature and develop a sound judgment.

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Anesthesia in Obstetrics -- Killer!

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Has any one of you ever had to tell a man that his wife or baby just died? That is a difficult and heart-breaking duty—it is even more so when you know that the death could have been avoided by foresight and care; as is sometimes the case when the death was due to anesthesia.

During the past few years, obstetricians have been justifiably pleased with the reduction in maternal and fetal mortality rates. However, it has taken this reduction in deaths due to hemorrhage, infection and toxemia to point up the alarming number of patients who die each year from anesthetic problems.

Many carefully conducted surveys during the past few years have indicated to all of us the importance of anesthesia as a cause of maternal mortality. It is necessary to mention the outstanding findings of only a few to impress upon you the serious problem we face in obstetrical anesthesia.

a. Penman[1] surveyed maternal mortalities from 1949 to 1952 inclusive in Philadelphia. Of all maternal deaths, 11-12% were directly due to anesthesia. In this 5 year period there were 22 anesthetic deaths:

12 with inhalation anesthesia
7 with spinal anesthesia
2 with caudal anesthesia
1 with pentothal anesthesia

These anesthetics were administered by the following personnel:

Staff Anesthetists	8
Nurses	8
Internes	4
Residents	2

The deaths were caused as follows:

Anoxia	8
Aspiration	5
Respiratory Paralysis with Spinal	4
Overdose of Spinal	3
Intrathecal Injection of caudal	2

b. Hellman and Hingson[2] state that anesthesia as a cause of maternal mortality is in 5th place behind hemorrhage, infection, toxemia and heart disease. In a review of 330 maternal deaths due to anesthesia in New York City, they found that 155 (47%) were the result of aspiration of vomitus, 24 were from overwhelming doses of a spinal anesthesia, 21

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were from caudal or continuous caudal anesthesia, and 119 from various other analgesic and anesthetic agents.

c. Newberger[3] found that 5% of all maternal deaths in downstate Illinois during 1948 to 1950 inclusive were due to anesthesia.

d. Fitzgerald and Webster[4] found that 44% of all maternal deaths in three St. Louis Hospitals were from anesthesia. Many of these cases had been listed under other causes of death.

e. Klein, et al[5], in a survey in the Bronx from 1940 to 1951, found that in the period 1940 to 1945 anesthesia was responsible for only 2% of maternal mortalities; but that from 1946 to 1951 it was responsible for 9% of the mortalities.

f. Lull and Hingson[6] point out clearly the depression of fetal respirations and delay in respirations of newborns after administration of ether for a period exceeding 15 minutes.

g. Acken[7] states that in 1245 caesarean sections there were 11 deaths, of which 8 (70%) were due to anesthesia.

Further recitation of statistics would only underline the importance of Anesthesia in Obstetrics as a Killer, and would convince us that no anesthetic agent or technic and no class of personnel is exempt from the dangers. So we will now discuss the **WHY** and **HOW** anesthesia causes these deaths, and **HOW** they can be prevented.

The basic causes of anesthetic deaths in obstetrics may be discussed under several main headings:

A. An obstetrical patient is usually unprepared or, at best, improperly prepared for an anesthetic:

1. The emptying time of the stomach late in pregnancy, and especially in labor, is prolonged to as much as 18-24 hours in most patients. This means we must assume that all obstetrical patients are candidates for aspiration of vomitus under anesthesia.
2. Labor usually begins unexpectedly and suddenly, with the result that many patients have eaten a heavy meal recently.
3. The patient in labor, and about ready for delivery, is emotionally disturbed and often uncooperative. Frequently the analgesics used have been given too late to produce a quieting effect on the patient such as one desires before starting an anesthetic.
4. Noise and bustle in the delivery room suite disturbs the patient.
5. Fear is common, especially in the younger patients and in those who have had difficulties in previous pregnancies.
6. Drugs may produce unfavorable reactions such as scopolamine delirium and narcotic respiratory depression.

B. Obstetrical patients tolerate anesthetic agents and technics differently from non-obstetrical patients:

1. All anesthetic agents are protoplasmic poisons, and

produce their desired effect only when used in subtoxic levels.

2. Agents used in spinal anesthesia can be given safely to a pregnant woman in only 1/3 to 1/2 their usual dosage for a non-pregnant individual.
3. The frequent occurrence of toxemia and anemia render many pregnant woman unusually susceptible to certain agents.
4. The labile circulatory system in pregnancy predisposes to sudden severe blood pressure drops.
5. Respiratory exchange at or near term is often considerably embarrassed by the distended abdomen.
6. The lordosis found at term increases the difficulty of lumbar puncture.
7. The increased intraspinal pressures during strong uterine contractions may force the anesthetic agent to a higher level than anticipated.

C. Inadequate equipment is often the result of:

1. The failure of the anesthetic department to check and service the equipment in the delivery rooms.
2. The shunting of old equipment to the delivery room.
3. The lack of adequate aspiration facilities for the adult.
4. No laryngoscope available.
5. No spare oxygen tanks and tools for changing them. Many new institutions have "piped-in" oxygen.
6. Lack of airways.
7. Lack of tray of emergency drugs—analeptic drugs, etc.

A recent survey of the more than 100 institutions in New York City where obstetrics is practiced showed that adequate equipment was **not** present in over 50% of the institutions. Many hospitals did not have gas machines or oxygen administering equipment in all their delivery rooms.

D. Inadequately trained personnel are often called on to administer anesthesia for delivery, especially at night, on week-ends, and holidays. It is also not uncommon for all trained personnel to be engaged in the operating room during the day. This means that internes, and unfortunately, completely untrained nurses administer dangerous agents about which they know little or nothing. Many are unfamiliar with the equipment, and more than one death has resulted from the administration of 100% nitrous oxide because the person using the equipment was not aware of an empty oxygen tank. Often an obstetrician will start an anesthetic and tell a nurse to hold the mask in place while he scrubs for the delivery. Such use of untrained personnel would not be tolerated for one minute in an operating room! Yet—we have obstetrical patients, who require more skill-

ful attention, receiving such treatment.

E. Carelessness is unforgivable at any time, but when a life may be at stake, it is criminal. Some acts of carelessness commonly encountered are:

1. Failure to check supply of oxygen.
2. Failure to remove dentures.
3. Failure to observe patient closely for signs of vomiting—especially with the mask clamped over the face.
4. The use of incompatible drugs—such as adrenalin or pituitrin when cyclopropane is being administered.
5. Failure to check pulse, blood pressure and respirations regularly when spinal is used.
6. Failure to have an intravenous solution running when spinal is used.
7. The anesthetist often leaves the delivery room before the patient has adequately reacted from her anesthetic.

The prevention of anesthetic deaths in obstetrics is the responsibility of three groups; (1) the hospital administration (2) the obstetrician and (3) the anesthesiologists and anesthetists. Until all three groups carefully survey their individual responsibilities and make corrections where deficiencies exist, we will continue to lose mothers and babies unnecessarily. I should like to point out some of the preventive measures which should be instituted or enforced.

The hospital administrator can:

1. Establish an adequately staffed anesthesia department headed by an anesthesiologist, or at least have an anesthesiologist available for consultation.
2. Make available adequate equipment for the delivery rooms.
3. Anesthesia coverage for delivery can easily be self-supporting and even remunerative.
4. Establish and enforce rules covering the types of anesthetics to be given by personnel of different qualifications after consultation with the directors of the anesthetic and obstetric departments.
5. **Note** is taken of the shortage of anesthetists in many areas, and this may well be a factor in some of the difficulties encountered.

The anesthesiologist has many difficulties to overcome in establishing adequate coverage for obstetrics, including shortage of anesthesiologists and nurse anesthetists, the irregular and often unpopular hours at which deliveries occur, and the lack of really ideal analgesic and anesthetic agents from both the maternal and fetal viewpoint.

Establishment of good obstetrical anesthesia service involves:

1. 24 hour coverage by adequately trained personnel—and I mean trained not only in surgical anesthesia, but also in the vagaries of obstetrical anesthesia.
2. Establishment of refresher training in the obstetrical aspects of anesthesia, since many anesthetists have considered this type of anesthesia as drudgery and an imposition, and so have side-stepped it as often as possible.

3. Establishment of extra remuneration for those working the night shift, week-ends and holidays.

4. Extra effort in proper training of obstetrical residents and anesthesia residents.

5. Further research into analgesic and anesthetic agents with due consideration given to fetal effects.

6. Indoctrination of all internes early in their hospital service in the simpler technics of obstetric anesthesia, in methods of infant resuscitation, and in the management of anesthetic emergencies.

7. Joint cooperation and conferences, with both obstetric and anesthetic departments participating, on obstetric anesthesia and problems of resuscitation so that many people are qualified to handle emergencies.

8. Understanding of the unpredictability of the time element in labor and delivery, so that less complaining will be done when an anesthetist loses extra sleep when called early. Unfortunately, this will continue to occur from time to time, since we have not yet acquired the ability to predict accurately the behavior of women—especially of women in labor.

The obstetrician has many responsibilities in reducing anesthetic deaths, some of which begin long before an anesthetic is ever started on a patient. Among these are:

1. Reappraisal of his own knowledge and use of obstetric analgesia and anesthesia.
   a. Careful selection of anesthetic to suit the individual patient's needs.
   b. Working knowledge of several types of anesthesia, their indications and contraindications, including both general and regional methods.
   c. Judicious use and withholding of analgesics.
   d. Patience in allowing the anesthetist to achieve the desired anesthetic level before beginning manipulations.
   e. Not allow himself to be stampeded into an unwise choice of anesthesia by a patient's desires.

2. Proper prenatal advice and training of his patients:
   a. Advise patient to avoid eating if labor starts or membranes rupture.
   b. Do not promise painless labor and delivery since this is as yet not possible in most cases with safety.
   c. Devote sufficient personal attention to each patient to obtain her confidence and cooperation.
   d. Do not promise beforehand which anesthetics or methods will or will not be used.
   e. Have patients in the best possible condition before the onset of labor by detecting and correcting, if possible, conditions such as anemia, renal, cardiac and pulmonary disorders.

3. Proper training for obstetric residents in the problems of obstetric anesthesia:

a. Each resident should receive careful instruction and supervision in the various technics of anesthesia under the department of anesthesia.
b. Residents must be allowed to use different methods of anesthesia under competent supervision just as they do different obstetrical procedures under supervision.

Summary

In summary, I submit that too many mothers die from obstetric anesthesia. I have pointed out where the responsibility for eliminating many of these unnecessary deaths lies, and how we can go about eliminating, to a great degree, obstetrical deaths from anesthesia.

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Sequelae of Endotracheal Intubation

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The evolution of endotracheal anesthesia has been a long and gradual one from Vesalius in 1542 to the present date. This development has been ably traced by Noel Gillespie in his monograph, "Endotracheal Anesthesia", first published in 1941.[1] Since 1941, however, the use of endotracheal anesthesia has become more and more a commonplace event. It is now, perhaps, a proper time to review just what can be accomplished by this modality and to call to mind what sequelae may be considered as disadvantages for this route of administration of gaseous anesthetic agents.

"Endotracheal anesthesia is a valuable technic which has profoundly modified both the patient's safety and the surgeon's freedom of access and asepsis and, hence, the anesthetist's peace of mind. Some enthusiasts have therefore attempted to use it as a "routine" in all patients and for every variety of operation. Nevertheless, its use necessitates a delicate operation, fraught with risks of trauma which vary inversely with the skill and experience of the intubator. Endotracheal anesthesia should always be available to any patient who may benefit from it: it should never be used without good cause."[1]

With these carefully chosen words in mind, let us consider what advantages may derive from the use of endotracheal anesthesia.

Advantages

To resort to primer methods for the sake of emphasis, one can accomplish only three things by the use of an endotracheal tube:

1. One can get substances into and out of the tracheo-bronchial tree with ease.
2. One can keep undesirable substances out of the respiratory tract.
3. One can, with safety to the patient, remove himself from the operative field, giving the surgeon unimpeded access to the surgical site.

To amplify on the first advantage: the obvious substances we pass through an endotracheal tube are oxygen and carbon dioxide, in the ordinary process of respiratory exchange, and the fixed anesthetic agent we may be using. This we can do in a nearly perfect fashion since we have eliminated the chief causes

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of upper respiratory obstruction: the tongue, the epiglottis and the vocal cords. It is also necessary, at least to a degree, to respire for the patient whom we have relaxed with curariform drugs or whose thorax has been opened and, by the use of an endotracheal tube, we can accomplish this without risk of distention of the stomach. Through the tube, we may pass a rapidly increasing concentration of anesthetic agent without reflex largyngospasm. While this enables us to deepen anesthesia more rapidly, it carries an inherent risk when the larynx cannot protect us against sudden lethal levels of irritant agents.

The substances we can get out through an endotracheal tube are accumulated secretions and the debris incident to such pathological conditions as bronchiectasis and other pulmonary infections. These appear to have a very real bearing on the incidence of post-operative atelectasis.[2] They are also important out of the operating room, where the anesthetist may be called upon to aid in the treatment of accumulated secretions in coma and other states with diminished activity of the cough reflex.

Our second advantage consists of the ability to keep undesirable substances out of the respiratory tract. We quickly think of gastric contents in this context. We are also familiar with the desirability of preventing blood, tissue fragments and dental debris from entering the trachea in operations where this is a possibility. It is not so obvious that we can keep the outside atmosphere from diluting the anesthetic mixture most perfectly with a well-fitting endotracheal tube.

The third advantage we gain from the use of endotracheal intubation needs little amplification. Our ability to withdraw from the surgical field when necessary is ably testified to by the increasing numbers of major operations undertaken in a leisurely fashion and with increasing safety in the region of the head and neck.

We can derive considerable satisfaction from the strides the specialty of anesthesia has made by virtue of these three advantages, but I doubt that we can accomplish much more than this by intubation. I doubt that relaxation is improved per se by this means. I also doubt that bleeding is lessened except insofar as oxygenation is improved and carbon dioxide excess eliminated. I should like to quote again from Gillespie: "The instrumentation of any orifice of the body is apt to engender minor traumatic sequelae, and the mouth, nose, pharynx, glottis and trachea are no exception."[1] That these maneuvers can result in sequelae other than minor ones, we shall now consider.

Disadvantages

Many of the sequelae of endotracheal anesthesia can be attributed to inexperience, insufficient relaxation or sheer awkwardness. The lacerated lip, the fractured tooth, the severe epistaxis are all examples much too familiar to those who attempt the instruction of others in the art of anesthesia. Moss colorfully tells us "... my early catheter introductions were along a very blood-bespattered path, slippery with mucus.... Ploughing up the pharynx with a laryngoscope in an attempt to dig out an epiglottis from a pool of bloody mucus is one of the least inspiring sights of modern anesthesia."[3]

These vicissitudes almost disappear as the anesthetist acquires experience and sometimes, skill. It is true that even in experienced hands an occa-

sional bizarre mishap can occur. Daly[4] reports one such occurrence where nasal intubation resulted in a submucosal dissection of tube along the posterior pharyngeal wall. However, there are two conditions which seem to have little bearing on the ability or skill of the anesthetist and which appear with sufficient frequency to cause us concern. These are granuloma of the vocal cords and subglottic edema.

Perusal of the literature gives us little idea of the true statistical incidence of these conditions.[5, 6, 7, 8] Several factors conspire in this. These are not common complications, and the experience of individuals is not of much value. Then, too, there is the habit of the patient of seeking another medical group when unforeseen complications occur post-operatively. This is particularly true of granulomata which takes a little time to cause symptoms. It is also why these lesions concern the ear, nose and throat specialist's disproportionate concern since this specialty treats most of them, a situation akin to the concern of the neurologist with the complications of spinal analgesia. However, these two complications occur frequently enough to constitute definite clinical entities, of considerable importance to the patient, and worthy of our careful consideration.

Granulomata of the Vocal Cords

The typical history of granuloma of the vocal cords is shown by the patient who leaves the hospital following an operation involving endotracheal anesthesia and gradually develops symptoms over the following three to six weeks. These symptoms may take the form of voice changes, chronic cough or other irritative phenomena or, less commonly, actual manifestation of mild respiratory obstruction.

For an understanding of this entity, we must consider the anatomy of the true vocal cords. The mucus membrane overlaying them is a delicate structure with little submucosa and poor blood supply. This results in a structure which, when traumatized, heals very slowly. The arytenoid cartilages to which the posterior end of the vocal cords are attached have a similar anatomy. The sensitivity of this region and the poor powers of repair which it possesses are not unlike the cornea of the eye. Thus, when the vocal cord is abraded by an endotracheal tube, a shallow ulcer appears, usually in the posterior two-thirds of the cord. As time goes by, indolent granulation tissue covers the bed of the ulcer and, eventually, piles up to a degree sufficient to cause symptoms in three to six weeks.

It is not quite clear why this trauma should be produced. According to Jackson,[6] core molds can be left in the larynx for several weeks without injury to the cords. We can do our utmost in prophylaxis by the use of tubes which are impeccably clean and free from irritating detergents. The surface should be glass-smooth. The size must not be excessively large, and gentleness in insertion is to be stressed. During maintenance of anesthesia, violent coughing, frequent swallowing and motion of the tube within the larynx is to be avoided. We should remember that the core mold in a larynx moves with the larynx and can safely be left in place for weeks. The endotracheal tube is fixed externally so that the larynx slides on it. This abrading action of a tube back and forth over the delicate vocal cords

may cause injury within a few hours.

When the granuloma has formed, surgical removal during direct laryngoscopy is usually indicated and results in a shallow ulcer, the precise lesion which started the whole difficulty. However, with complete voice rest until healing has occurred, fortunately, the formation of a secondary granuloma can usually be avoided.

From the patient's point of view, after he has apparently entirely recovered from one operation, he finds himself faced with another hospital admission and another surgical procedure. This is succeeded by a fairly prolonged period of absolute voice rest, which is usually economically disabling. He may fail to see the connection between a pilonidal sinus and his larynx. His complaints are to the surgeon and usually not to the anesthetist. Such a surgeon, not very surprisingly, becomes somewhat less than enthusiastic about the use of the endotracheal route for his future patients.

Subglottic Edema

The above entity, granulomata, seem to concern themselves almost entirely with the adult patient. Subglottic edema, which we will consider next, is, on the other hand, almost exclusively seen in children. The age incidence parallels that of croup or laryngo-tracheo-bronchitis, being more common in infancy and early childhood.

In the typical case, after an endotracheal anesthesia which is usually somewhat prolonged, the child reacts with nothing more alarming than some huskiness of voice and, perhaps, a dry, barking cough. As the hours go by, increasing signs of respiratory obstruction are seen. There is inspiratory stridor, retraction in the suprasternal region, with an occasional hoarse, non-productive cough. As the condition worsens, the pulse accelerates, and restlessness and anxiety become marked. If therapy is not successful, cyanosis appears, deepens, and death may occur. This condition reaches a maximum in from twenty-four to thirty-six hours and usually subsides quite rapidly.

Again, a consideration of anatomy helps us to understand this entity. Eckenhoff has ably described the differences between the infantile and the adult larynx.[9] The chief peculiarity of the infant larynx which interests us is the fact that its narrowest cross-section occurs, not at the glottic chink, but at the level of the cricoid cartilage, the sub-glottic region. This region has the added peculiarity of an abundant submucosal layer which, practically speaking, has the characteristics of erectile tissue, such as the nasal turbinates. These two factors are of great interest to us. Due to the essentially funnel-shaped larynx of the infant, we can frequently pass an endotracheal tube with ease through the vocal cords only to find that obstruction is met a very short distance beyond, at the level of the cricoid cartilage. Should we traumatize this region by the use of force, it will respond, as all tissues do when injured, by swelling. Mathematically speaking,[9] if we produce one millimeter of edema in the cricoid region of the adult with its 20 millimeter diameter, the cross-section area is reduced only 19%. However, the same degree of edema occurring in an 11 pound infant with a cricoid diameter of 4 mm.[10] results in a reduction of cross-section area of 75%. Thus, we see that trauma which might go unnoticed in the

adult can be lethally asphyxial in the infant.

Gentleness in intubation with a tube of a carefully selected size is definitely indicated. No force whatsoever should be used to overcome resistance met in the subglottic region. The question of the role of infection in the production of subglottis edema remains an unsolved one. At the present time, it would seem prudent to use scrupulously cleaned tubes with a minimum of handling of the portions which will enter the larynx and trachea. Then, too, since time seems a definitely important factor, a certain burden is laid on the shoulders of the surgeon to accomplish his task expeditiously in this younger age group.

Watchfulness in the first postoperative twenty-four hours is of the utmost importance. An atmosphere of high humidity is probably the best therapeutic agent we have available. A croupette is usually indicated routinely for the younger child with a prolonged endotracheal anesthesia. The older pediatric patients may be watched carefully for premonitory signs, at which time, continuous cold steam atmospheres should be inhaled.

The crucial decision one has to make is whether to tracheotomize or not. This is a dramatically lifesaving measure if it is not unduly postponed. To wait for the appearance of frank cyanosis is to find one's self operating on a moribund child, in frantic haste, and with a needless mortality. Extreme caution should be used with sedation in these cases. It is a great temptation to quiet the restlessness of anoxia with sedatives. It may also be a certain way to subdue the respiratory drive which is keeping them alive. These patients may truly sleep their lives away with injudicious sedation.

Discussion

This paper is in no way meant to be an indictment against endotracheal anesthesia. It is merely an attempt to point out that there are certain inevitable complications attendant upon its use, two of which are discussed in detail. The author will continue to employ this route in the future extensively, as he has done in the past. However, it will always be with the thought in mind that certain penalties will be paid from time to time, and that the risks must be carefully weighed against the gains.

Summary

1. The endotracheal route for administering anesthetics can accomplish only three things:
   a. One can get substances into and out of the tracheo-bronchial tree with ease.
   b. One can keep undesirable substances out of the respiratory tract.
   c. One can, with safety to the patient, remove himself from the operative field, giving the surgeon unimpeded access to the surgical site.

2. There are certain sequelae associated with this route almost unavoidably. Two complications are treated in detail.

3. Granulomata of the vocal cords and sub-glottic edema are considered from the viewpoint of symptomatology, anatomical considerations, prophylaxis and treatment.

4. A plea is made for weighing the advantages and the penalties of this valuable anesthetic practice.

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Treatment of Hypotension when Blood Replacement is Ineffective

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The use of blood transfusions has greatly lessened the incident of severe shock in patients undergoing surgery. On occasions, however, we still see a patient in severe shock who fails to respond to blood therapy. It is the purpose of this paper to review some of the possible reasons for failure of transfusion therapy and some of the measures said to be useful when such therapy fails.

Much new information about the pathophysiologic changes in shock has come from the research laboratories, but the mystery of this syndrome still seems far from solution. Shorr, Zweifach and associates[10,27] have demonstrated by means of direct visualization of the terminal vascular bed that there are three phases of the shock state: an initial compensatory phase characterized by vasoconstriction, augmented vasomotion, and increased responsiveness to epinephrine; a transitional stage in which the hyperreactivity gradually diminishes, and a decompensatory "irreversible" stage in which vasomotion is absent and blood collects in the capillaries. When the change from "reversibility" to "irreversibility" takes place, or why the change occurs, is not known.[8] Among the factors that have been suggested as contributing to deterioration of circulation following hypotension are the presence of circulating humoral factors presumably produced by renal and hepatic ischemia, overactivity of the autonomic nervous system, and overwhelming infection associated with tissue anoxia.[10] At present, few clinical applications of these hypotheses have been made, and in the clinical management of shock, emphasis is still placed on the prevention and on early energetic treatment while the state is still reversible.

Fundamental treatment of hemorrhagic or traumatic shock, it is generally agreed, consists of rapid restoration of circulating blood volume with whole blood. Vasopressors such as norepinephrine have also been found to be of value in some cases as an adjunct to blood replacement, but opinion differs as to the physiologic soundness of the use of vaso-

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pressors in shock therapy.[8,10] When the irreversible stage of shock develops, administration of blood only serves to accentuate the derangement of circulation as it increases congestion in the capillaries, and vasopressors are of no value in restoring the activity of the terminal vessels.[27] Moore,[10] however, points out that the term "irreversible" is a defeatist term which should never be used in the clinic, and says that we should "remind ourselves at frequent intervals that severe and apparently fatal shock, if of short duration, is readily susceptible to reversal by appropriate treatment." When the fundamental therapy fails, as it sometimes does, we are, therefore, forced to consider as possible reasons for the failure some error in total management of the case, or the need for some therapeutic measure other than blood.

Correction of Errors in Management

Probably the first thing that should be thought of when faced with failure of transfusion therapy is that some technical error or some omission in management of the case is the causing factor. It has been said that, in the absence of active sepsis, failure to respond to transfusion therapy may be taken as evidence of inadequate replacement or continuing blood loss.[5,8] This is undoubtedly true in the majority of cases and the adequacy of replacement therapy should be the first consideration when a patient does not evidence the expected result from transfusion of whole blood.

Estimation of what constitutes "adequate" replacement is a difficult problem, especially in cases where bleeding has been profuse. Far too often, estimation of blood loss is based on guess-work, and it has been shown that the ability to estimate blood loss by appraisal of the sponges and drapes results in errors ranging from 50 per cent to 200 per cent.[1] Since the hazard of overreplacement is as great as that of underreplacement, it would seem that some more reliable method should be used. Formulas based on the degree of reduction of systolic blood pressure, on hemoglobin and hematocrit may offer a rough guide to the reduction in blood volume, but are not too reliable.[1,5] Studies showing average blood loss for various operative procedures have obvious limitations as a means of estimating how much blood should be given in an individual case. The most practical and simple method for estimating blood loss with reasonable accuracy is that described by Bonica and Lyter[1] in which sponges are weighed and the blood in the suction bottle is measured. In calculating amount of blood, 1 Gm. is considered as 1 cc. with no correction for the slight difference of the specific gravity of blood from unity. Saline used for sponges is measured and subtracted from the total. The margin of error with this method has been shown to be less than 5 per cent. Since this method does not entail much additional work on the part of operating room personnel, it seems that it should be used whenever extensive surgical procedures are contemplated or when the patient's condition increases operative risk.

It is also possible that some error in the management of anesthesia may contribute to the failure of homeostatic mechanisms and thus hinder the response to blood replacement. For example, a deep level of anesthesia, regardless of the agent used, can depress the compensatory mechanisms of the peripheral vascu-

lar bed. Anoxia and carbon dioxide excess may contribute to the deterioration of peripheral circulation. Unfortunately, the method used to provide adequate pulmonary ventilation may itself contribute to the existing circulatory depression. Motley,[19] Maloney,[16] and Hubay[15] have shown that artificial ventilation with intermittent positive pressure can significantly reduce cardiac output when circulation is normal. In patients suffering from circulatory depression, Maloney[16] found that the use of intermittent positive pressure could cause a fall in blood pressure and cardiac output of sufficient magnitude to threaten the life of the patient. It has been shown that the use of a negative pressure phase to produce expiration effectively counteracts the reduction in cardiac output,[15,16] but devices for administration of positive-negative pressure are not yet available for general use in connection with anesthesia apparatus. Until such equipment is generally available, it would seem that intermittent positive pressure should be employed with great caution in the patient who is in shock, and that meticulous attention be paid to maintaining the mean mask pressure as low as possible. The only way this can be accomplished when applying intermittent positive pressure by use of manual compression of the breathing bag is to allow an expiratory phase of at least one and one-half times the duration of the inspiratory phase (and preferably longer), and to make certain that the bag is not over-inflated at any time, so that during expiration the pressure can fall quickly to atmospheric pressure as soon as the inspiratory pressure is released.

When it seems reasonably certain that blood replacement has been adequate and no technical mismanagement is responsible for the patient's failure to respond to blood replacement, one must look elsewhere for the cause of failure. A review of the recent literature suggests three possible reasons which may account for the failure of blood transfusions. These are acute adrenal insufficiency, myocardial depression, and complications due to the transfusion itself such as citrate intoxication and transfusion reaction.

Acute Adrenal Insufficiency

The possibility that acute adrenal insufficiency may be the cause of a patient's failure to respond to blood and vasopressor therapy has received increasing attention recently. There seem to be two aspects of this problem to consider: (1) acute adrenal insufficiency precipitated by the stress of surgery in the patient who has adrenal disease, and (2) the possibility of a temporary adrenal cortical insufficiency resulting from circulatory impairment in shock.

Adrenal Insufficiency in the Patient with Organic Disease

Hotchkiss,[14] Hayes[12] and others have called attention to the possibility of acute adrenal insufficiency developing in patients not previously known to have adrenal or pituitary disease. The difficulties in diagnosing adrenal insufficiency in the borderline cases are pointed out by Hayes.[12] Since a patient in adrenal crisis may not survive unless proper therapy is instituted, the possibility should be kept in mind when unexplainable shock develops or persists in spite of administration of blood and vasopressors.

Root[22] says that there seems to be a fairly typical course that should arouse suspicion of adrenal cortical insufficiency. The most important sign is severe circulatory collapse,

which occurs suddenly, is not related to blood loss, and which fails to respond to blood transfusions and vasopressors. According to Root, the time of occurrence may be shortly after the operation or up to 1 to 2 days later. Moore[10] and others report cases in which adrenal insufficiency developed during the course of operation. In the conscious patient, additional signs are mental confusion, disorientation and coma.[22]

Certain laboratory tests may be used to confirm the diagnosis of acute adrenal insufficiency. Hotchkiss says that, in the presence of marked hypotension, low blood serum sodium, low serum chloride, and low carbon dioxide combining power indicate adrenal failure. He emphasizes, however, that the diagnosis of hypoadrenalism cannot be rejected if normal serum sodium and chloride levels are found because the hemoconcentration may mask the actual loss of total serum electrolytes.

The eosinophil count may also be used as a diagnostic aid. There is considerable variation in the findings of different investigators in regard to the eosinophil response to surgery, but there seems to be agreement about the general pattern of the response. There is an initial rise at the beginning of the operation, then a prompt and rapidly progressive fall.[12] The eosinophil count may reach zero and is usually below 20 as compared with a preoperative average of 150.[22] The degree of eosinopenia correlates with the severity and length of operation, and usually remains low for 1 to 2 days. A high or normal eosinophil count in the presence of shock during the first 24 hours postoperatively would be a strong indication of adrenal insufficiency.[22]

Because the laboratory tests are not of great reliability, Hayes suggests that in some case a test dose of adrenal cortical extract (50 cc.) may be necessary to determine the presence of adrenal insufficiency.[12] If adrenal insufficiency is the cause of the circulatory collapse, there should be a prompt, though transient, improvement in blood pressure and quality of the pulse in response to the test dose.

Prediction of Adrenal Cortical Insufficiency Pre-operatively

It has been advocated that, because of the seriousness of acute adrenal insufficiency, a test of adrenal function be administered as part of the routine preoperative preparation of a patient.[11] Unfortunately, there is not a simple, reliable test that can be used to screen these patients with border-line adrenal insufficiency.[6,13,14,22] Eik-Nes and associates[6] have demonstrated that the measurement of plasma levels of 17-hydroxycorticosteroids for a six-hour period following the intravenous administration of 25 I.U. ACTH reliably measures adrenal cortical activity and is not subject to variations by abnormal function of some other organ, but this test may be a procedure beyond the scope of some laboratories. As yet, therefore, it does not seem practical to include a test of adrenal function as part of routine preoperative preparation, although it would be helpful to do so for those patients who are to undergo extensive surgical procedures.

Until such time that there is a simple effective test available for determining a patient's capacity to withstand stress, we must rely on empirical judgment, and Hotchkiss' description of the types of patients who may be prone to develop acute

adrenal insufficiency during or following surgery is useful for alerting us to the possibility of this complication. Patients in whom one might suspect hypo-adrenalism are: (1) those subjected to an extensive surgical procedure; (2) senile patients with chronic debilitating disease; (3) psychotic patients; (4) patients who have been treated with cortisone, hydrocortisone, and/or corticotrophin; (5) patients who have urological disorders. Hotchkiss points out that, in this last group of patients, injury to the adrenals may occur during renal surgery, and that disease of the genito-urinary tract, such as carcinoma or tuberculosis, may involve the adrenals.

Treatment of Acute Adrenal Insufficiency

Since the problem we are considering is that of the patient in whom adrenal insufficiency has not been suspected, discussion of treatment is limited to active therapy. The treatment of acute adrenal insufficiency has three basic purposes: (1) administration of fluid and electrolytes to restore and maintain the volume and composition of extracellular fluids; (2) administration of glucose to combat hypoglycemia, and (3) administration of adrenocortical steroids.[9]

Hormonal Therapy—Of primary importance is the rapid administration of adrenocortical hormones. The preferred hormone is hydrocortisone.[9,22] Goodman and Gilman suggest a dose of 50 mg. given by rapid infusion, followed by a maintenance infusion at the rate of 10 mg. per hour until the patient recovers from the signs of crisis. Root advises administration of 100 mg. in 500-1000 cc. of fluid given at the rate of 10-20 mg. per hour. In addition a dose of cortisone may be given intramuscularly in several depots for later effect. Cortisone is administered in doses of 50-75 mg. every six hours until the patient is out of danger.[22]

Five to 10 mg. of desoxycorticosterone acetate is usually also administered in the treatment of Addisonian crisis. DCA may predispose to fluid retention and pulmonary edema, and since hydrocortisone and cortisone exert some influence on sodium balance,[9] it may be advisable to rely on these hormones when treating adrenal insufficiency during the course of operation.

Fluid and Electrolyte Therapy—Fluid, electrolyte and glucose requirements can be met by administration of glucose in isotonic saline solution.[9,14] Great care must be exercised to avoid giving too much fluid and salt because of the danger of precipitating pulmonary edema. This is particularly important in the patient with poor cardiac reserve, since in such patients, hydrocortisone and cortisone can promote edema formation.[9,14] It is recommended that fluid volume should be limited to 3500 cc. in 24 hours.[9]

Vasopressors—Concomitantly with the administration of hydrocortisone, a vasopressor may be administered to maintain blood pressure until the the hydrocortisone is effective.[14,22] Hotchkiss suggests 6 cc. of 1 per cent neosynephrine or 4 mg. of norepinephrine in 1000 cc. 5 per cent glucose, given through a second vein. The rate of administration should be determined by the blood pressure response and should be sufficient to keep the systolic blood pressure above 100 mm. Hg. Hotchkiss states that in most cases it is possible to discontinue the vasopressor within four hours after initiation of hormonal therapy.

Temporary Adrenal Cortical Insufficiency

The question of whether adrenal cortical steroids are of value in the treatment of shock from hemorrhage or trauma in patients who do not have organic adrenal disease has been extensively investigated and remains a highly controversial subject. Several investigators have postulated a form of adrenal insufficiency which is functional or temporary in nature. One hypothesis is that excessive demand for hormones exceeds the capacity of the adrenal cortex to produce them and the gland is thus exhausted.[11,12,14,22]

Hayes[12] suggests that a state of temporary adrenal insufficiency may occur in shock as a result of impairment of blood flow through the adrenal vascular bed. It has been demonstrated that in the initial compensatory phase of shock blood flow through the kidneys is reduced, this reduction being partly due to the fall in arterial blood pressure but also to increased resistance, or vasoconstriction, in the kidneys. From this fact, Hayes postulates a probable reduction in adrenal blood flow which would result in a reduction in amounts of adrenocortical steroids available per unit time for peripheral tissue utilization.

Hayes[11,12] studied eosinophil responses during the course of operation in patients with various endocrinological disfunctions and also in patients undergoing major abdominal operations who were presumed to have normal endocrinological function. He reports that in cases where a severe stress was imposed, the eosinophil count rose. The elevation in eosinophil count correlated with a fall in blood pressure or with sudden blood loss. In one case, an estimated blood loss of 500 cc. required replacement with 1500 cc. to stabilize the blood pressure and eosinophil count, whereas in another case, both were stabilized with 500 cc. of blood and administration of aqueous adrenocortical extract.

There is evidence that adrenal cortical hormones are essential to the function of the homeostatic mechanisms of the peripheral vascular bed. Zweifach has shown that normal tone, vasomotion, and reactivity of the terminal vascular bed is dependent on normal adrenal cortical function.[10] There is also evidence that the response of the peripheral vascular bed to vasoconstrictor substances is dependent on the integrity of the adrenal cortex.[25] Williams and associates[26] found that cortisone increases vessel tone in the normal animal, but found no evidence of increased responsiveness to norepinephrine when cortisone was given to normal animals.

Many investigators, however, do not feel that there is any convincing evidence that the adrenals can fail because of excessive demand.[8,22] Evidence has been presented that adrenal function remained unimpaired in fatal shock and in moribund patients dying from various causes.[22] It has also been reported that administration of adrenal cortical hormones have failed to affect the course of experimental shock. Zwiefach states that their findings indicate that the irreversible phase of hemorrhagic shock is not associated with failure of adrenal secretory activity and that the hyporeactive state of the peripheral vascular bed was not improved by the administration of either cortisone or ACTH.[10] Howard,[10] and France and Jennings[7] report no beneficial effects from the use of cortisone in experiments on dogs in hemorrhagic shock[10] or in

shock of non-hemorrhagic origin.[7] However, France and Jennings report beneficial effect from the use of cortisone in 5 patients in a series of 25 patients suffering from shock of varying etiology. It is interesting that in two of these patients the eosinophil count was low; in the others it was high.

In view of the highly contradictory ideas about the possibility of a functional or temporary adrenal insufficiency being associated with shock, and the inability to demonstrate the usefulness of adrenal steroids in the treatment of experimental shock, there seems to be no justification at present for the use of adrenocortical hormones as an adjunct to blood replacement in the "routine" treatment of shock. The fact that there is now available a convenient intravenous preparation of hydrocortisone may tempt us to resort to its use in situations where the conventional measures against shock would suffice, so it is advisable to remember that there is an even more compelling reason to employ caution in the use of hydrocortisone than the inability to demonstrate its effectiveness in experimental shock. The metabolic effects of the adrenal corticoids are complex and as yet not fully understood and their side-effects are numerous. Some of the side-effects that should particularly be considered when the use of these drugs is contemplated in the surgical patient are the tendency to lower resistance to infection,[9,24] the possibility of delayed wound healing,[9] and the tendency to produce hypercoagulability of the blood.[9,18] Thomas, who warns against indiscriminate use of cortisone and ACTH, suggests that their use be reserved for "occasions of peril" and he limits occasions of peril to the threat of death or prolonged or permanent incapacitation.[24]

Until more is known about these dangerous and complex drugs, it would seem prudent to refrain from the use of adrenocortical hormones during the course of anesthesia and surgery except in those cases where acute adrenal insufficiency can be demonstrated or is strongly suspected, and in those "occasions of peril" when the patient's condition is critical and other measures have failed. Under such circumstances, it seems justifiable to resort to the use of hydrocortisone even when it is not possible to confirm a diagnosis of acute adrenal insufficiency.

Myocardial Depression in Shock

In traumatic or hemorrhagic shock, the mechanism of circulatory failure is generally assumed to be peripheral in origin. The reduction in cardiac output is initiated by the decline in venous return, and the consensus is that no defect in the heart's function contributes to the establishment of "irreversibility." There seems to be, however, evidence that in some circumstances failure to respond to transfusion therapy may be due to myocardial failure precipitated by the shock state.

Wiggers has shown that in irreversible shock in dogs blood replacement will cause a rise in venous pressure without improvement in cardiac output, and suggested that myocardial failure may be the reason for the failure to respond to transfusion. Davis reports that in dogs that have died from circulatory failure due to reduction in blood volume, the heart is frequently found to be lacking in tone and even dilated.[5] Moore points out that the coronary circulation is a terminal vascular bed, and that when the terminal vascular bed as a whole is involved in the pathologic

changes of shock, it is reasonable to assume that the myocardium is suffering along with the other tissues of the body.[10] There is evidence that myocardial ischemia occurs following hemorrhage in elderly patients,[8] but there is conflicting opinion about its being a factor in the failure of response to blood replacement in the young.[8,17] Master and coworkers[17] reported the occurrence of 59 cases of acute coronary insufficiency among 103 patients who had suffered acute hemorrhage, and concluded that hemorrhage is a frequent precipitating cause of coronary insufficiency. In this series, some of the patients had no history of antecedent heart disease. Sharpey-Schafer[10] describes a syndrome associated with the loss of small amounts of blood over a long period of time in which the venous pressure is high, cardiac output increased, and hemoglobin, low. Transfusion given at this time may result in pulmonary edema and death from what appears to be heart failure.

Measures that have been suggested as beneficial in cases in which there is reason to suspect that myocardial depression may be contributing to the lack of response to transfusion therapy are intra-arterial transfusion and digitalization.

Intra-arterial Transfusion

There have been clinical reports that intra-arterial transfusion resulted in marked improvement in cases where intravenous transfusion produced congestion in the presence of continued low arterial blood pressure.[8] The success of arterial transfusion in such cases has been attributed to the fact that perfusion pressure is increased in the coronary circulation. On the other hand, laboratory investigation has failed to reveal any superiorty in the effect of the intra-arterial transfusion as compared with intravenous transfusion.[3,8] Case and associates[3] showed that coronary flow is not benefited by arterial transfusion, and believe that arterial transfusion is more effective than intravenous transfusion only in mitral stenosis, cardiac arrest, or in cases where cardiac contraction is almost completely ineffective.

Digitalization

The general opinion seems to be that there is no justification for the use of digitalis in shock therapy unless there is definite evidence of congestive heart failure. Davis, however, reports that administration of digitalis in experimental hemorrhagic shock resulted in an elevation of blood pressure ranging from 10-40 mm. Hg.[5] Sankey and Crawford[23] and Powell[21] have reported the use of digitalis as an adjunct to shock therapy in anesthetized patients, and both reports state that beneficial results were obtained. Powell reports one case in which digitalization appeared to improve the response to intra-arterial transfusion.

Powell believes that digitalization should be resorted to before the conventional signs of cardiac decompensation are apparent, and feels that early digitalization is justified in the anesthetized patient because the heart subjected to the potential strains of anesthesia, surgery, and blood loss is not a "normal" heart. Most anesthetics tend to produce some degree of myocardial depression, and anoxia and carbon dioxide retention adversely affect the heart muscle. Other factors such as extreme position and trauma impose strain on the heart. It seems reasonable that the heart subjected to such strain may have a diminished reserve and may not be

able to meet the additional strain of peripheral circulatory failure.

Signs which are considered an indication for digitalization when response to blood therapy is poor are (1) venous congestion, (2) progressively diminishing pulse pressure, (3) tachycardia (Powell finds that the pulse is often slow, however), (4) cold, but dry skin, and persistent cyanosis which is not relieved by administration of 100 per cent oxygen. Sanky and Crawfod also consider that inability to maintain a satisfactory plane of anesthesia without producing severe circulatory depression is an indication for digitalization.

The appearance of any of these signs in a patient in whom decreased myocardial reserve might be suspected should arouse suspicion of myocardial depression. Patients in whom decreased myocardial reserve might be present are the aged, those with cardiovascular or pulmonary disease, and those with a history of repeated hemorrhage. In addition, Powell believes that patients who develop exaggerated circulatory depression following small doses of depressant drugs may have diminished myocardial reserve.

The choice of digitalis preparation used for rapid digitalization appears to depend on personal preference. According to Goodman and Gilman, all digitalis preparations have the same type of myocardial action and all have approximately the same margin of safety.[9]

Complications due to Transfusion Therapy

Another factor which may account for the failure to respond to blood replacement is the transfusion itself. Two complications which seem to be particularly associated with the use of multiple transfusions are citrate intoxication and massive hemorrhage due to coagulation defects.

Citrate Intoxication

Recent reports have called attention to the danger of citrate intoxication when multiple transfusions are administered rapidly. The toxicity of citrate with its depressant effects on the heart is well known, but early studies showed that there was a wide margin of safety with its use as an anti-coagulant in blood. When small transfusions are used, or moderately large amounts of blood are given slowly, no apparent harmful effects are observed. However, when citrated blood is given rapidly and in large amounts, the toxic action of citrate on the heart is evidenced. Nakasone and coworkers[20] emphasize that the hazard of citrated blood may be greater in patients who have myocardial disease or conduction abnormalities, and Cookson[4] believes that even small transfusions of citrated blood should not be used for patients undergoing cardiac surgery. It may also be possible that the heart subjected to reduced circulating blood volume is more sensitive to the effects of low serum calcium levels.[2] This has been demonstrated in dogs subjected to graduated blood loss, and Bunker feels that it may also be true of the human heart. The possibility of citrate intoxication is increased in patients with liver disease,[2,20] or with mechanical obstruction to hepatic circulation.[2] It is also possible that the hazard of intoxication is increased in conditions that impair the metabolic processes, such as barbiturate anesthesia, nutritional deficiency, and possibly hypothermia.[4]

Bunker and associates[2] found that ionized calcium levels remained within normal limits when moderately

large volumes of blood were administered at a rate of 500 cc. in 30 minutes, but when it was administered at a rate of 500 cc. in 15 minutes, the concentration of serum citrate rose above safe levels and there was a tendency for the calcium level to fall.

There is apparently no outstanding clinical sign of citrate intoxication. The development of tetany during rapid transfusions has been reported, but it did not occur in Bunker's series even when the ionized calcium level was very low. Bunker feels that profound circulatory collapse or general anesthesia may mask tetany. The low calcium levels do not seem to be responsible for increased bleeding.[2] Electrocardiographic changes are typical of those associated with hypocalcemia produced by other causes,[20] but without electrocardiographic evidence and serum calcium studies, apparently the only sign of citrate intoxication is the presence of a persistent hypotension when multiple transfusions have been administered rapidly.[2]

The treatment usually recommended is the administration of a soluble calcium salt, and some writers have suggested the use of calcium gluconate as a preventive measure against citrate intoxication whenever large volumes of blood are to be given. The dosage recommended is 3 cc. of 10 per cent calcium gluconate for every 500 cc. of blood. Bunker and associates used calcium chloride in doses of 1-10 Gm. and report that their results were not too satisfactory. They found it difficult to estimate the proper therapeutic dose of calcium and emphasize the danger of elevating the serum calcium to toxic levels. They believe that when large quantities of blood must be administered rapidly that it is better to avoid the use of citrated blood than to rely on treatment with calcium salts, and suggest such preparations as decalcified blood (with which the calcium dosage can be accurately estimated), packed red blood cells or resuspended red blood cells. When such preparations are not available, the cautious use of a calcium salt is apparently the only recourse.

Massive Hemorrhage due to Coagulation Defects

Coagulation defects which result in uncontrollable bleeding may be caused by reaction to incompatible blood[18] or may be due to lack of clotting factors in banked blood.[2] The administration of multiple transfusions appears to increase the risk of transfusion reaction and there seems to be evidence that blood that is compatible in the major blood groupings may contain undetermined or unknown incompatible factors. The hazard of reaction to incompatible blood is particularly great in the anesthetized patient, since none of the usual early signs are apparent. Lack of expected improvement in circulation seems to be about the only early sign of transfusion reaction, and this sign is one subject to various interpretations. Increased oozing and the appearance of mottled cyanosis probably indicate that a rather large amount of incompatible blood has been given.

Little is known about the mechanism of the change in the coagulation process precipitated by the incompatible transfusion. McKay and associates[18] studied the changes in coagulation factors produced by incompatible blood transfusion and concluded that there appeared to be two basic causes of the hemorrhagic state: (1) afibrinogenemia or fibrinogenopenia and (2) the development of a heparin-like anti-coagulant.

They recommend that when uncontrollable bleeding develops an immediate estimate of all the main clotting factors be obtained. If afibrinogenemia is shown to exist, purified fibrinogen should be administered. If the presence of an anti-coagulant factor is suspected, then an anti-heparin agent such as toluidine blue should be administered.

Summary

The problem of the anesthetized patient in severe shock who fails to respond to blood transfusions has been discussed. Rapid restoration of blood volume by whole blood is the basic treatment of hemorrhagic and traumatic shock. In the search for the cause of failure to respond to transfusion therapy the first consideration should be to ascertain the adequacy of blood replacement, and to correct any technical errors in surgical or anesthesia management which may contribute to depression of circulation.

Other possible causes for failure to respond to blood transfusion are acute adrenal insufficiency, myocardial depression, citrate intoxication, and transfusion reaction. These are uncommon complications, but because of their seriousness, the anesthetist should be alert to the possibility of their occurrence so that warning signs are not overlooked. It should be emphasized that, in the light of present knowledge of the shock syndrome, adrenal cortical steroids and digitalis are not recommended in the treatment of hemorrhagic shock unless there are reasonably clear-cut indications for their use.

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Personnel Relations

E. G. Chuinard, M.D. *
Portland, Oregon

For a few moments I would like to appear in the official position of president-elect of the Oregon State Medical Society to extend to the members and friends of this association an official greeting and commendation from the physicians of Oregon. This state and this city, and particularly those of us whose work it is to provide medical services in this area, are highly honored at having this institute come to Portland. No one, unless it be yourselves, should be more keenly interested in the educational efforts you are making through these institutes than the physicians who so greatly depend upon your being a part of the surgical team that provides care for our patients. It is therefore with sincere appreciation and respect that the physicians of this state welcome all who are attending this institute, and wish for you a most pleasant and profitable sojourn with us.

Ever since the demonstrations of ether anesthesia by Long in 1842 and Morton in 1846 there has been a continual increase in the variety of anesthetic agents and methods of administration. The development of agents and technics of anesthesia has kept pace with, and helped make possible, advancements in other fields of medicine. Of all the fields of medical practice, anesthesia has been most closely aligned with surgery and the brilliant advances in surgery have been made possible to a large extent by the no less spectacular advances in anesthesia.

The fact that ether was the first surgical anesthesia developed is perhaps one of the most fortunate things that happened in the history of anesthesia. Ether provided a margin of safety that permitted the physician to induce anesthesia in the patient and then leave its continued administration to the nurse while he left the head of the table to perform the surgery. From this, it was but a short step to having the nurse relieve the surgeon entirely of anesthesia administration, except for assuming the responsibility. No example in the annals of the physician-nurse relationship so exemplifies the degree of confidence bestowed by the physician —and earned by the nurse—as does the development of the nurse anesthetist. And as new anesthetic agents and modes of administration have developed, the nurse anesthetist has kept pace.

Since ether anesthesia was developed by a physician and a dentist in an effort to provide a means of relief from human suffering, it in-

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evitably followed that physicians should continue to pursue, by research and clinical application, their interest in the field of anesthesia. As is true of other drugs, all anesthetic agents carry with them certain side effects that may be potentially dangerous. The new avenues of administration which have been added to the respiratory route—intraspinal, intravenous, regional—have added to the complexity of anesthesia. In addition, complicated and extensive modern surgical procedures have created new demands. All of these factors have placed a greater responsibility not only upon the anesthetic, but upon the anesthetist. It is trite, but true, to observe that the efficiency and safety of the anesthetic is determined more by the one who administers it than by the nature of the agent itself. The array of modern anesthetic agents, and the responsibility these place upon the anesthetist requires, in many instances, the presence of a physician who has a knowledge of anatomy, physiology and pharmacology beyond that provided by the training of a nurse anesthetist. The advance of medicine in all fields has made specialization in anesthesia necessary. Such specialization now includes some 3000 physicians who are devoting their practice exclusively to anesthesia.

A perusal of published accounts of your previous institutes shows that the entrance of the physician into the field of anesthesia posed for the nurse anesthetist one of her most vexing problems. This is indicated by questions such as: "What is the proper relationship of the nurse anesthetist to the anesthesiologist?" or, "Is the nurse anesthetist on the way out?"

Every problem that comes to us, however, has its silver lining if for no other reason than it serves to stimulate a critical review of our own position. Such objective reappraisal of ourselves usually tends to illuminate not only our virtues, but also our shortcomings. It is natural for us to react emotionally to those who seem adversely critical and unappreciative of us. Usually, however, certain salutary benefits result, and this can be said to be gratifyingly true of the nurse anesthetist.

You have become more pridefully conscious of your own history and contributions as you have emphasized these to others. While engaging in recruitment programs, you have resold yourselves to yourselves. In conducting statistical surveys you have discovered that instead of there being a diminution of your place in the field of anesthesia, there has been an increase, not only in your numbers, but in the demands for your services.

Recent figures show that there now are 106 schools for the training of nurse anesthetists as compared with 52 in 1948. More important than the number of schools and the number of graduates, however, is the quality of instruction provided by the schools and the caliber of the persons who graduate. That these high standards will be maintained is in part assured through your establishment, in 1951, of a system of accreditation for your training schools, and through your allowing only graduates from accredited schools to become members of the American Association of Nurse Anesthetists. The prestige you are gaining as a result of your efforts toward better training and service is an added inducement for nurse anesthetist graduates to strive to meet your standards and qualify for membership in your association. If I were a nurse anesthetist I most certainly

would desire and seek the privilege and distinction of being a member of the A.A.N.A.

A review of your JOURNAL shows that certain subjects have recurred persistently in the forum discussions in your institutes indicating that these subjects are the problems uppermost in your minds. The consistent theme which runs through all of these problems is a concern for the status of the nurse anesthetist in the hospital, and her relationship with the hospital administration, the patient, the nurse, the surgeon, the anesthesiologist, and the law.

In the past years you have had expert opinion and advice with regard to the various facets of these relationships. In reviewing the published accounts of these forums what impressed me was the fact that no clearly established rule and order consistently delineates these relationships throughout our country. Much of the discussion has consisted of a review of experiences peculiar to a certain locale, or to the procedure that has developed in that locale.

It is easy to understand that the legal implications for the nurse anesthetist will vary according to the statutes of different states. I will not essay the role of legal advisor, except to comment that legal implications are becoming more important to all who are concerned with medical service. Whereas, in the nostalgic "good old days," it was unquestioned for nurses to give ether anesthesia, today both the nurse and the physician may need to be more circumspect with regard to the privileges and responsibilities delegated to the nurse anesthetist.

Because the nurse anesthetist is usually an employee of the hospital, the hospital is probably the most influential determiner of the environment in which she serves. If a physician is in charge of the anesthetic department of the hospital, the intimate relationship in which he is placed with the nurse anesthetist probably becomes the greatest factor in determining the efficiency and pleasantness of her work. The anesthesiologist can provide a constant source of supervision, instruction, and reassurance for the nurse anesthetist —a factor which constitutes no real change from the historical physician-anesthetist relationship in the past. This brings forth the observation that it is not merely the inadequacy of a nurse anesthetist's training that limits her competency in some extreme situations in modern surgery, it often is the relatively equal incompetency of the operating surgeon to instruct and supervise her.

I have tried to indicate that the role of the anesthesiologist is important on two counts: 1) As a direct participant in the surgical team in difficult cases where his knowledge of anatomy, physiology and pharmacology is important for the best possible anesthesia; and 2) as a participant in the training and supervision of nurse anesthetists. As a surgeon who is interested in the best possible service to my patient, I would not set up one factor as being more important than the other. I am happy to say that I work in a hospital where the relationship between anesthesiologists and nurse anesthetists is one of assistance to one another with resultant benefit to both the surgeons and the patients.

As a surgeon I feel on more secure ground in making observations about the relationship of the nurse anesthetist and the surgeon. I suppose it is as humanly natural for certain surgeons to feel more at ease with certain anesthetists as it is for some

patients to feel more at ease with certain nurses and physicians. Personal attributes, as well as technical skill, contribute to the facility of the surgical team. The anesthetist and the surgeon should contribute to each other's ease, because such rapport will be sensed by the patient and will contribute to his confidence.

May I suggest that the surest way for the nurse anesthetist to put the surgeon at ease and to secure his confidence is to keep him informed about the patient. The anesthetist—and for that matter the anesthesiologist—should never keep a flutter of anxiety about the patient's condition to herself with the idea that she will not disturb the surgeon. The surgeon will remain at greater ease if he can rely on the anesthetist to give him all pertinent information promptly. Certainly the surgeon should be consulted about the use of drugs to be used, as might be indicated by the patient's changing condition. It is not that the nurse anesthetist might not be able to make such decisions herself on the basis of her experience and her knowledge of the patient as she sits with his pulse and blood pressure and respirations literally at her fingertips, but it is simply that through training and licensure and contract with the patient the responsibility belongs to the surgeon.

We have referred to the rapid changes that are occurring in the array of anesthetic agents, and their modes of administration. These have added to the problems of the surgeon and the anesthetist. The use of intravenous anesthetics, and the use of adjunct drugs like curare, and the use of procedures like intubation, places all of us squarely on the horns of the dilemma: should a nurse anesthetist be permitted to administer these drugs and procedures? On the other hand, should an anesthesia be entrusted to a nurse anesthetist who cannot exercise some proficiency in this regard? Again, there is no universal rule. Cautious experience will probably provide the best guide as it did in those early years when nurses first began dropping ether. A nurse anesthetist's training and supervision, and her own good attitude and good sense, will in most instances indicate to what extent she will use the anesthetic agents and procedures at her disposal. The exercise of such good judgment always has been, and ever will be, the essence of the professional attitude.

We have referred to the ease and confidence which is a necessary part of the surgeon-anesthetist relationship. As better training has gradually given each of us more self assurance, and there has been accompanying changes in the decorum of both the surgeon and the anesthetist, good relationships have been better able to develop. The surgeons and anesthetists who once performed their duties almost through sheer courage alone are rapidly disappearing from the modern surgery taking with them the belligerency that always accompanies bluff. Another requisite for confidence in ourselves—in addition to excellent training—is a willingness to prudently confine ourselves within the scope of our training. Respect from others comes to us when we voluntarily limit our activities to those practices which are within the boundaries of our training and ability, be we nurse anesthetist or surgical specialist.

All members of the surgical team should take care that the mutual confidence and reliance which each places in the other because of expert scientific training does not overshadow humility. The training of our head

and hands must be tempered by the warmth of our hearts. The public relations committee of the Oregon State Medical Society formulated the phrase, "Our patients will not care how much we know if they do not know how much we care." The same remark is applicable to all members of your association. Those patients who surrender their consciousness to you will probably give little thought to what your training may have been, but they will surely remember the kindness you showed them and the confidence you evoked in them.

I have purposely placed little emphasis on rules and regulations and procedures that may define and assure your future place in the medical team. The facts referred to at the beginning of this paper are ample proof that you have the training and the program that provides you with the tools which will make your place secure.

Sometimes I think we physicians also become too contemplative about whether the public can get along without us, when what we need to make sure of is that the public can get along with us. We must be certain that in our drive to develop our abilities we do not disproportionately emphasize the scientific aspects of our profession without regard for those things which mark the "human touch."

Sometimes I regret the advent of sodium pentothal anesthesia. Patients now come demanding that they be given "that anesthetic in the vein." It used to be that the patient who had had surgery before would request "that same anesthetist" for the next operation. Now he slips off into unconsciousness without a chance to get acquainted with the anesthetist—that is, unless a part of the armamentarium of the anesthetist includes a disposition to keep alive that personal equation known as "the human touch."

In conclusion, let me remind you that you have every right to be proud of your profession's history of service. No longer is there need for you to be on the defensive—if, indeed, there ever was. You are making a noble effort to assure your continued place among the medical services. May I suggest that along with your scientific training you continue to emphasize a proportionate human and kindly coddling of your patients for the greatest assurance of your future place.

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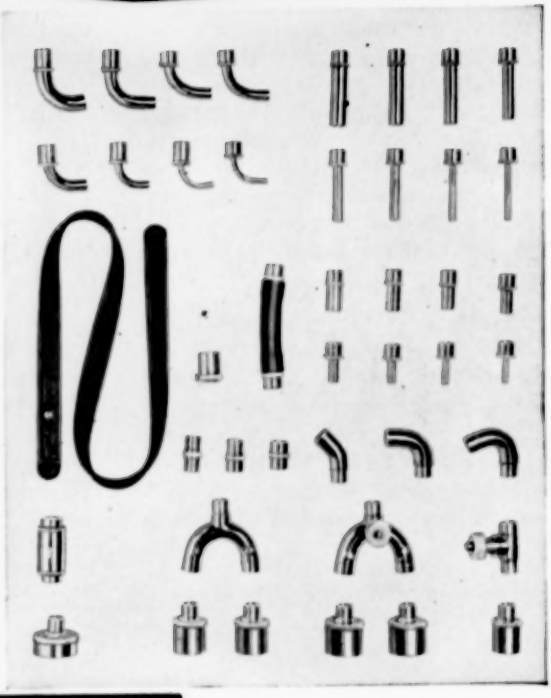

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Abstracts

Fabian, L. W., Ling, P. C., Ewing, Paul and Shafer, C. W.: Further experimental and clinical evaluation of a new syncurine antagonist. South. M. J. **48**:251-259 (March) 1955.

"As a basis for the present investigational work we must refer to our previous work with the antagonism of Syncurine (Decamethonium Bromide) by drug No. 49-204. The initial experimental work has been published and more recently clinical evaluations have been presented. With this foundation, our concepts of myoneural physiology and certain relationships of respiratory and cardiovascular mechanisms to the end plate responses have gradually become altered or further substantiated. The primary phases of investigation have stimulated our interest in determining the mechanism and sites of action and certain other problems of interest and importance. Concerning the clinical data, we have accumulated approximately 50 well controlled cases in which certain aspects involved in the antagonism of Syncurine were demonstrated. However, no authentic case of 'prolonged Syncurine depression' has occurred; therefore we can only assume the efficacy of this new antagonist in such an actual entity.

"From the experimental and clinical data accumulated, a number of conclusions and/or speculations are presented: (1) It has been demonstrated that normal to moderately high arterial carbon dioxide levels may be essential if prolonged respiratory depression is to be avoided. (2) The efficiency of antagonism between Syncurine and drug No. 49-204 increases during hypercapnia and decreases during hypocapnia. Supervening hypoxia will, of course, render such resuscitation more difficult and it should be emphasized that oxygen consumption and utilization may be impaired in either state depending on the severity of deviation of the carbon dioxide as compared to normal. By progressively decreasing the arterial content of carbon dioxide during hyperventilation, oxygen dissociation (at least in vitro) shows a proportionate decrease which could well provoke tissue hypoxia. (3) On the basis of the results obtained, we believe the site of action of both Syncurine and its antagonist to be dual, the former acting peripherally for the most part, the latter apparently exerting some degree of influence on the respiratory center. The mechanism would seem to be one of competitive action at both myoneural and medullary levels. As mentioned previously, however, we feel confident that for its greatest response, drug No. 49-204 is to a significant extent dependent on its synergistic activity with carbon dioxide.

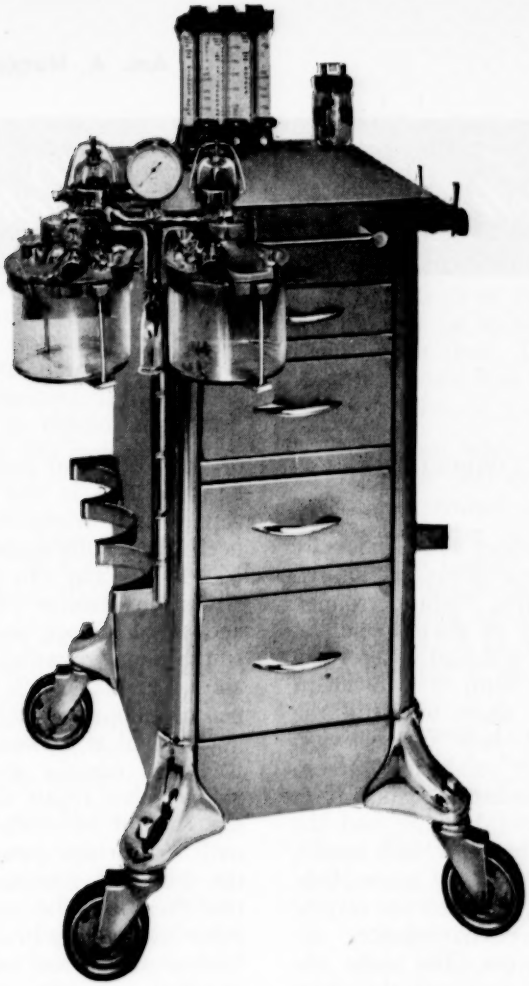
"(4) Certain properties of specific antagonism might well be those relative to similar molecular configu-

ration, e.g. Syncurine—drug No. 49-204, and morphine N-allylnormorphine. The derivatives not only act as antagonists, but when used alone or in excess of the agent, may and usually do provoke a depressor response although not so marked. Drug No. 49-204 resembles weak curare in its activity. (5) The clinical value of these findings is as yet indefinite. However, evidence has been presented in which respiratory depression and peripheral muscle weakening may be prolonged by hyperventilation and hypocapnia. The reverse is also true up to a certain point; this pertains to carbon dioxide narcosis at higher levels of content. Of course, hypoxia is to be avoided and assisted respiration during such depression is mandatory. Nevertheless it should be remembered that hypocapnia is without a doubt a major contributory factor in prolongation of respiratory and peripheral muscle relaxation. By allowing development of a level of arterial carbon dioxide content commensurate with the control level, reports of prolonged apnea may well be of the past. Hypoxia, during this 'waiting period' (or period of apnea without assisted respiration) is usually quite easily detected by noting the typical changes associated with cardiovascular responses to oxygen desaturation or unsaturation. Any change in pulse rate or rhythm should ordinarily be construed as a manifestation of hypoxia during the above mentioned 'waiting period.' This occurrence demands greater attention to provision of adequate ventilation."

Davis, D. A.: The role of curare in anesthetic deaths. South. M. J. **48**:37-38 (Jan.) 1955.

"In a recent publication Beecher and Todd presented statistical data to show that death occurred in one patient in every 370 who had been given curare or a curare-like drug. In contrast, the mortality rate in patients who did not receive curare was one in 2,100. This evidence was based on a study of 599,500 anesthetics given during a five-year period in ten university hospitals. The res ipsa loquitur conclusion derived from these figures might be that curare is an intrinsically lethal drug and that its use should be curtailed sharply if not abandoned entirely. This conclusion was not drawn directly by the authors but has been so interpreted by many surgeons. The validity of the above data cannot be disputed easily, but there is little doubt that exceptions will be taken to the conclusion suggested above, and it is probable that in the near future other statistics will be submitted which will present curare in a much more favorable light. . . . But the fact remains that considerable suspicion has been raised in the minds of both the medical and lay public, and it is certain that in the future curare will be subjected to detailed scrutiny, particularly when death follows its use. Perhaps the term 'curare death' may relieve some of the burden borne at present by the much abused and ill-defined term 'cardiac arrest.' If so, it will add nothing to our understanding of why anesthetized patients die. It is probable that the mechanism of death due to curare is the same in the operating room as in the jungles of South America. Whether the drug is given by a sterile needle or poisoned arrow, curare kills primarily by interfering with the ability of the respiratory muscles to move oxygen into, and carbon dioxide out of the lungs. Death is not necessarily sudden in either situation. Minutes or

(Continued on page 289)



CABINET MODEL 460 SERIES

The Model 460 Series is mounted in a beautiful four drawer Stainless Steel Cabinet, with large ball bearing casters. Embodying INDIVIDUALLY CALIBRATED FLOWMETERS, as well as Tank Pressure Gauges, Reducing Regulators, Yoke Shut-Offs, Lung Pressure Gauge, Built-in Aspirator, as well as an efficient CO_2 Absorber and Ether Vaporizer.

Price of Three Gas Unit as shown **$735.00 FOB Chicago, Ill.**

COMPLETE STOCK OF ENDOTRACHEAL ACCESSORIES

PORTEX TUBES **INFLATABLE CUFFS**

Chicago Anesthetic Equipment Company

1662 WEST OGDEN AVENUE **CHICAGO 12, ILLINOIS**

Write for complete information and name of your authorized dealer

Notes and Case Reports

50 Cent Conductivity

Conductive rubber soled shoes are available in some areas in limited styles to operating room personnel, but the majority of doctors, nurses, and anesthetists still find it necessary to rely on some form of attachment to regular duty shoes to fulfill the requirements of safe conductivity. The black rubber conductive attachments, the aluminum band from bottles of parenteral fluids, and the aluminum strip in gauze face masks, are all effective means of accomplishing conductivity, but there are several objections and inconveniences attached to their use. The main objection is the comparatively short life of the attachment and the necessity of frequent checking to confirm that one is still conductive. Some are very difficult to attach comfortably, others discolor the shoes and hose badly, and all are very conspicuous.

Copper and aluminum have long been recognized as two metals possessing a very excellent degree of conductivity. Both are incapable of producing a spark when struck against another hard object. Since these two properties are the basic requisites for accomplishing safe conductivity of electrical energy, either metal is a preferred material for grounding purposes.

The method of grounding shoes to be described is one which utilizes copper nails driven through the shoe heel. These nails range in length from ½″ to 2″ and can be obtained at almost any lumber yard for an average of $1.35 per pound. (A pound suitable for grounding purposes averages 50 nails, which makes the cost per nail approximately 3c.)

One nail is needed for each shoe heel and can be installed by any modern shoe repair shop at an average cost of 50 cents per pair. The nails are driven completely through the shoe heel with one end touching the floor on the outside and the other end coming in contact with the bottom of the heel on the inside. If installed properly, they cause no discomfort, and are adequately effective for the life of the shoe. The one disadvantage of this method of conductivity is that the chemical reaction between perspiration, body heat, and copper produces a green discoloration of the heel of hose.

Our hospital uses a stat-o-test machine to determine conductivity, and shoes with the copper nails consistently register 13 amperes. (On this particular machine, 7 is considered highly satisfactory.) There is no appreciable variation regardless of whether the shoe soles and heels are leather, non-conductive rubber, or plastic, and the nails can be put into

flat, cuban, or wedge heels. Although we used two nails in each heel on the first tests, one nail apparently is fully effective.

Although I did not originate this idea, I have become so enthusiastic about it that I am anxious for everyone to have the opportunity to utilize it. If any interested person encounters difficulty in obtaining the nails, I will be happy to obtain and mail them at cost, to any person, group, or hospital.

PEGGY STEWART
R.N., M.A.A.N.A.
Midland, Texas

The above note was submitted to Harriet L. Aberg, A.A.N.A. Representative to National Fire Protection Association's Committee on Hospital Operating Rooms for her opinion. Miss Aberg's reply follows:

"One of the objections mentioned in the first paragraph is 'the necessity of frequent checking to confirm that one is still conductive.' This must be done with any type of conductive shoe or shoe appliance. Wax, dirt, etc. can insulate the head of the copper nail just as effectively as it can insulate conductive soles and any of the shoe appliances.

"The objection of conspicuousness of some attachments is in my opinion an advantage.

"If the surface of the only point of conductivity on a shoe is a nail head of approximately 1/4" or less, then there is no assurance that it will be in contact with the conductive floor at all times.

"Anesthetists, as well as others in operating rooms, often hook their heels on the rungs of the stools with the foot, or toe, so that the ball and heel sections of their shoes are in contact with nothing but air. Often (especially in the absence of a footstool) they have just the tips of the toes on the floor. In both of these cases, they would not be in contact with the conductive floor. The presence of the nail could lead to a false sense of security.

"If those who use the copper nail in the heel are aware of the extremely limited area of conductivity, and are careful always to keep it in contact with the floor, then this would seem an excellent idea. During an anesthetic, however, it would be difficult to keep this in mind at all times."

HARRIET L. ABERG
A.A.N.A. Representative to
National Fire Protection
Association's Committee on
Hospital Operating Rooms

N.F.P.A. STANDARDS FOR OPERATING ROOMS

After a brief outline of what static is (See May, 1955 Journal, Page 127) and why anesthetists are interested in its hazardous aspect, perhaps scrutiny of N.F.P.A. pamphlet No. 56 would be in order. This is a standard which is put out by the National Fire Protection Association through its Committee on Hospital Operating Rooms. The standard is divided into three parts,—General, Construction and Equipment, and Administration. There is also an Introduction and an Appendix.

The first section of part I, GENERAL is concerned with the Nature of Hazards with subheadings—Com-

bustible Anesthetic Agents, Combustible Disinfecting Agents, Electrical Systems—Shock and Spark Hazards, Electrostatic Spark Discharges, Flames and Hot Materials.

As the Appendix explains, There is cognizance "of suggestions that the detonation of ether peroxides formed by the oxidation of ether over a period of time may be a cause of explosions in anesthesia machines." Because of this all anesthetic departments should include in their safety rules some provision for emptying and cleaning all ether vaporizers every day. Most of our vaporizers are made of clear glass which allows light to come in contact with ether, and light facilitates the break-down of ether. Heat also facilitates the break-down of ether and our anesthetic apparati can become heated by standing in the sun or left near heating units in a room. These are the reasons for taking the precaution of emptying vaporizers every day. The ether can be put in a safety can or returned to the can in which it came, tightly corked, and reused when needed, or poured on a window sill or some outside surface to evaporate. If ether is disposed of by evaporation, it must be done in an area remote from any source of ignition, remembering that ether vapor is heavier than air.

In the subheading Electrostatic Spark Discharges, the insulators include nonconductive casters on furniture, nonconductive tires on stretchers, nonconductive stool tips, nonconductive soles of stockings and shoes; in other words anything which would insulate persons and objects from each other, assuming the floor is conductive.

The material in the other subheadings of part I are self explanatory.

Construction and Equipment

Having discussed static and its importance in the explosion picture and briefly explained Part I General of the National Fire Protection Association standard No. 56, "Recommended Safe Practice for Hospital Operating Rooms", we will next consider the sections in Part II—Construction and Equipment. This takes up the greater portion of No. 56. It starts by defining "Hazardous Locations". These locations include "storage locations" and "anesthetizing locations". It should be remembered that operating rooms, delivery rooms, anesthesia rooms, corridors, utility rooms, accident rooms, treatment rooms, any place where combustible anesthetic agents are to be used are "anesthetizing locations" and considered hazardous locations. It is in this section that the height of the "hazardous locations" is defined as extending to "5 feet above the floor" in anesthetic locations.

Next is the section on Ventilation. Included are ventilation requirements for "storage locations" which are mandatory, and "anesthetizing locations" (operating rooms, etc.). Safety standards for mechanical ventilation and air-conditioning units are given and the Appendix for this section carefully explains the intent of the safe practices recommended.

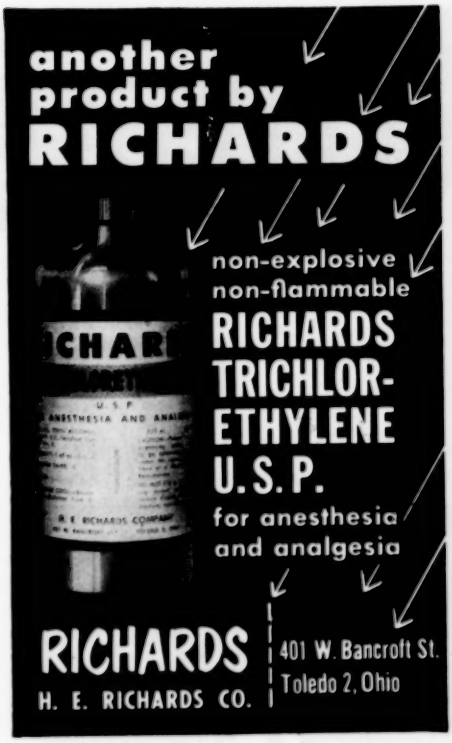
Section 4 on Storage and Piping of Gases is rather specific in mentioning piping **only** for nonflammable medical gases and other inert gases, never for combustible anesthetic gases. There is provision for proper placement of shut-off valves in these pipelines to insure greatest patient safety. Yoke construction on anesthetic apparati is mentioned and the Appendix explains the Pin Index Safety System.

Electrical Wiring and Equipment is the subject of section 5 and the specification of an ungrounded electrical system is contained here with the Appendix supplying the interpretation. Electrical wiring for each area, "storage and anesthetizing locations", is spelled out. The arrangement of circuits, transformers, signal lights, resistance values, and all the details of the ungrounded system are given. This is a most inclusive section and the points covered also include safety requirements for Service Equipment, Equipment Grounding, Control Devices, Receptacles and Attachment Plugs, Lighting Switches, Lighting Fixtures, Signaling Systems, Portable Equipment (this includes suction, pressure or insufflation equipment, x-ray and high frequency equipment), Low Voltage Equipment and Instruments.

"Reduction of Electrostatic Hazard" is the last in Part II and the Appendix for this section explains the requirements for conductive flooring. The method of testing conductivity of floors, furniture, mattresses and pads, rubber tubing and parts, plus the conductivity requirements of these, is given in section 6. The hazards of mechanical intercouplers are explained.

It wouldn't be amiss for the anesthetist to go over Part II with the chief engineer of the hospital. Greater understanding would surely result with better cooperation in the maintenance of high safety standards in operating rooms with regard to flammable anesthetics.

HARRIET L. ABERG
A.A.N.A. Representative to National Fire Protection Association's Committee on Hospital Operating Rooms



(Continued from page 284)

hours may pass before the effects of inadequate ventilation appear. Only recognition of death is sudden. . . . Curare was introduced as an adjunct to good anesthesia. It was never intended that it be used as a substitute for good technics of anesthetization. One of the principal criteria by which the ability of an anesthetist is judged is his or her ability to induce unconsciousness easily and smoothly without visible alteration in the circulatory and respiratory processes of the patient. This is known as the 'art' of anesthesia and is not learned easily. . . . It would seem reasonable to direct attention from curare to those who abuse the privilege of use of this drug."

Legislation

Emanuel Hayt, LLB., Counsel, A.A.N.A.

By means of this photograph, we are pleased to introduce Mr. Emanuel Hayt to those members who may not have had an opportunity of meeting him in person. Mr. Hayt is legal counsel for the American Association of Nurse Anesthetists and is well known in the hospital field as an authority on the law of hospitals and hospital personnel.

ATTORNEY GENERAL OF WEST VIRGINIA HOLDS NURSE ANESTHESIA LEGAL.—The secretary of the Medical Licensing Board of West Virginia asked the state attorney general the following question:

> "Is it permissible for a nurse to administer endotracheal and spinal anesthesia under the statutes governing the practice of medicine and nursing in this state?"

In reply, the attorney general on June 10, 1955 quoted a section of the West Virginia law which reads as follows:

> "In any case where it is lawful for a duly licensed physician and surgeon . . . to administer anesthetics, such anesthetics may lawfully be given and administered by any nurse who has been duly registered as such under the laws of this state: provided, that such anesthetic is administered by the nurse in the presence and under the supervision of such physician or surgeon."

The all-inclusive language of the phrase "in any case" must necessarily include, the attorney general said, the authority for registered nurses to administer endotracheal and spinal anesthesia.

Book Reviews

Selection of Anesthesia. By John Adriani, M.D., Director, Department of Anesthesia, Charity Hospital; Professor of Surgery, School of Medicine, Tulane University of Louisiana; Chief Clinical Professor of Surgery and Pharmacology, School of Medicine, Louisiana State University, New Orleans, Louisiana. Buckram. 327 pages, 35 illustrations. Springfield, Illinois: Charles C. Thomas, Publisher, 1955. $6.50.

This book, consisting of 36 chapters, is designed for persons who must select anesthesia and the author stipulates that the book is to be used in no way as a source of information for the technic of anesthesia.

Much of the information that appears in the authors' previous publication **The Pharmacology of Anesthetic Drugs** is here transferred to a specific clinical application. Each group of anesthetic agents is discussed in considerable detail in relation to its clinical use. More than half of the book is devoted to this section. The second section of the book develops the relationship of non-surgical diseases to anesthesia. Diseases that may complicate the surgical procedure are developed according to body systems and according to specific diseases and extremes of age.

In the third section of the book, the type of operation and its relation to the selection of anesthesia is presented. It is in this section that the author's vast clinical experience seems best to be translated into the purpose of the book. Here, as in the second section, each sub-section is developed according to body systems. The book is well indexed.

Saddle Block Anesthesia. By Ray T. Parmley, M.D., Director, Department of Anesthesia, St. Francis Hospital; Lecturer in Anesthesiology, University of Kansas School of Medicine; Staff Anesthesiologist, St. Joseph Wesley Hospitals; Consultant, Veterans Administration Hospital; Wichita, Kansas. Pajco Lexide. 58 pages, 9 illustrations. Springfield, Illinois: Charles C. Thomas, Publisher, 1955. $2.50.

One of the American Lecture series, a monograph on the American Lectures in Anesthesiology, this small book is prepared by an author who worked with Dr. Adriani in the development of this particular technic. Nurses who work with doctors using this technic—particularly in obstetrics—and teachers in schools of anesthesia, will find this an excellent reference for information concerning this procedure.

Basic Sciences in Anesthesiology (A Guide for Study). By Arthur B. Tarrow, Lt. Col., United States Air Force (Medical Corps); Diplomate, American Board of Anesthesiology; Fellow, American College of Anesthesiologists; Assistant Professor of Anesthesiology, Graduate School, University of Texas; Chief of Anesthesiology Section, Lackland Air Force Base, San Antonio, Texas. Paper. Ed. 2. 274 pages. San Antonio, Texas: Lydette Publishing Co., 1955.

This paper bound book was prepared by Lt. Col. Arthur B. Tarrow of the United States Air Force as an aid to persons who are preparing for the examination of the American Board of Anesthesiology. Following the five major divisions of the American Board of Anesthesiology examinations in Basic Science, the author

has divided each of these into three subdivisions. The first subdivision is an outline of the contents; the second is a bibliography; and the third is a series of questions that have appeared in the examinations. For each question a reference is given so that the person who is studying from this manual will have the source readily available. Originally copyrighted for use in the U.S. Air Force School headed by Dr. Tarrow, requests for copies became so numerous that the Air Force found it impractical to continue to distribute the book. Permission was granted for private publication. The book's 274 pages are mainly devoted to questions and their answers.

The Principles and Practice of Surgical Nursing. By D. F. Ellison Nash, F.R.C.S., Assistant Surgeon, St. Bartholomew's Hospital, London; Senior Surgeon, Children's Hospital, Sydenham; Consultant Surgeon, Chailey Heritage Hospital; Lecturer in Surgery, Demonstrator of Practical Surgery, St. Bartholomew's Hospital Medical College; formerly Hunterian Professor, Arris and Gale Lecturer, Royal College of Surgeons; Examiner in Surgery, Queen's University, Belfast, and in the Faculty of Dental Surgery, Royal College of Surgeons. 1007 pages, 371 illustrations. London: Edward Arnold (Publishers) Ltd,. 1955. $7.00.

Upon first glancing through this book it seemed to this reviewer that it probably was one of the many books on nursing that would have relatively little of value for the student or graduate nurse anesthetist. However, on closer scrutiny, it developed that a great portion of the book would seem to be of immense value. Such chapters as those on hemorrhage, shock and fluid balance, artificial respiration, care of the unconscious patient, postoperative care, antiseptics, preoperation preparation, the use of oxygen, specific chapters on operations of the thorax and other systems of the body, postoperative complications, and others should be of immeasurable value to nurses in the field of anesthesia. The chapter on anesthesia itself contains more valuable information than is often found in publications on the subject of anesthesia. Although there would be a slight adjustment of the information to allow for the differences in equipment and regional practice, this book is wholeheartedly recommended to all nurses who are learning, those who are teaching, and those who are administering anesthesia.

Pain—Its Mechanisms and Neurosurgical Control. By James C. White, M.D., F.A.C.S., Chief of Neurosurgical Service, Massachusetts General Hospital, Associate Professor of Surgery, Harvard Medical School; and William H. Sweet, M.D., F.A.C.S., Associate Visiting Neurosurgeon, Massachusetts General Hospital, Associate Clinical Professor of Surgery, Harvard Medical School; with assistance in the psychiatric sections of Chapters IV and X from Stanley Cobb, M.D., Former Chief of Psychiatric Service, Massachusetts General Hospital, Bullard, Professor of Neuropathology, Emeritus, Harvard Medical School; and Frances J. Bonner, M.D., Assistant in Psychiatry, Massachusetts General Hospital and Harvard Medical School, Boston. Buckram. 736 pages. 134 illustrations. Springfield, Ill.: Charles C. Thomas, Publisher, 1955.

Although this book will not be of primary interest to nurse anesthetists, its vast collection of valuable information will be a source for better understanding by those who are in the field of pain relief. To teachers in the field of anesthesia this book will also serve as an excellent reference. Six years in preparation, the book is extremely authoritative, well indexed, and contains an extensive bibliography.

Classified Advertisements

NURSE ANESTHETIST: 140 bed suburban hospital under direction of M.D. Attractive salary. Delaware County Hospital, Drexel Hill, Pennsylvania.

NURSE ANESTHETIST: Female. Accredited modern 200 bed hospital. Department directed by anesthesiologist, assisted by three nurse anesthetists. Pleasant working conditions, hours, paid vacation, social security, group insurance and retirement plan. Excellent staff of surgeons. Good salary and merit increases. Apply: May Aileen Davies, M.D., La Crosse Lutheran Hospital, 1910 South Avenue, La Crosse, Wisconsin.

REGISTERED NURSE ANESTHETIST: 135 bed general hospital in charming southern city of 18,000 short drive from from Gulf of Mexico. Well qualified surgical staff. Salary range $380 - $416 per month commensurate with experience. 4-weeks vacation with pay, sick leave, 2½ day weekend every 4th week. Apply: Administrator, John D. Archbold Memorial Hospital, Thomasville, Georgia.

TWO NURSE ANESTHETISTS: Modern well equipped hospital, 75 beds, 25 bassinets. Comfortable living quarters - TV in living room; town of 8000, 50 miles from Philadelphia, near Wilmington, Delaware. 5-day week, $450 per month and full maintenance. About 100 operations per month. Apply: Charlotte W. Ager, Superintendent, Union Hospital, Elkton, Maryland.

NURSE ANESTHETIST: For 400 bed Joint Commission Accredited hospital. Salary $425.00 per month and meals. 40-hour week. Many employee benefits. Apply: Dr. R. Weyl, Anesthesiologist, Mount Sinai Hospital, Chicago 8, Illinois.

WANTED: Nurse anesthetist. 450 bed general hospital, fully approved. Salary open. Apply: Administrator, McKeesport Hospital, 1500 Fifth Ave., McKeesport, Pennsylvania.

NURSE ANESTHETISTS WANTED: Immediate openings at modern 327 bed general hospital with active up-to-date anesthesia department. Registration not a requirement if sufficiently experienced. Pleasant working conditions. Attractive salary in addition to complete maintenance. Generous vacation, sick leave and holiday allowances. Unusually good opportunity for those seeking permanent positions in a well established department. Apply: Director, Eastern Maine General Hospital, Bangor, Maine.

REGISTERED NURSE ANESTHETISTS: 40 hr. week, permanent positions open for surgery and obstetric departments. Liberal vacation and sick leave policies. Social security, overtime pay, extra pay for night duty. No call duty. Automatic pay increases. Apply: Chief Nurse Anesthetist, Harper Hospital, Detroit 1, Michigan.

NURSE ANESTHETIST: For approved general hospital. Must be able to administer latest types anesthetic agents. Salary $350.00 to $375.00 per month plus full maintenance. Annual vacation and sick leave. Retirement benefits if desired. Apply: Administrator, Robinson Memorial Hospital Ravenna, Ohio.

ANESTHETIST: Registered nurse. Modern 200 bed general hospital. Forty hour week. $435 to $487.50 per month. Paid vacations and sick leave. Overtime on liberal case basis. Medical anesthesiologist in charge. Apply: Chief of Department of Anesthesia. Pontiac General Hospital, Pontiac, Mich.

WANTED: Surgical Anesthetist for 150 bed general hospital central Nebraska. Excellent working conditions and personnel policies. $450 per month and full maintenance. Apply: Box M-28, Journal American Association of Nurse Anesthetists, 116 S. Michigan Ave., Chicago 3, Ill.

THIRD ANESTHETIST wanted in approved general hospital of 184 beds in city of 25,000. Regulated hours and good working conditions. 4-weeks paid vacation. Paid sick leave. Salary according to qualifications. Minimum $400.00 per month plus full maintenance. Living accommodations in nicely furnished Nurses' Home. Apply Trinity Hospital, Minot, North Dak.

NURSE ANESTHETIST WANTED: 500 bed teaching hospital on campus of state university. Starting salary $4512.00 with regular increases, cumulative sick leave, paid vacation. Apply to anesthesiologist in charge, University of Virginia Hospital, Charlottesville, Virginia.

WANTED: Two Nurse anesthetists, 300 bed hospital, attractive employment conditions; starting salary $400 per month. Night and weekend calls taken in rotation every fourth night and every fourth weekend. Six paid holidays annually. Minimal amount of obstetrical anesthesia. Full maintenance provided if desired. Approximately 5000 anesthesias administered annually for all types of surgical cases. Department is under supervision of an anesthesiologist. Apply to J. M. Schwab, M.D., Chief of Department of Anesthesiology, Geisinger Memorial Hospital and Foss Clinic, Danville, Penna.

NURSE ANESTHETIST: Oral surgeon in the vicinity of Boston, Mass. would like nurse anesthetist beginning August 15, 1955. Leonard S. Fox, D.D.S., 69 B. Court St., Westfield, Mass.

NURSE ANESTHETIST: 165 bed General Hospital, fully approved. Annual vacation and sick leave. Salary open. Apply Administrator, W. A. Foote Memorial Hospital, Jackson, Michigan.

NURSE ANESTHETIST: 600 bed approved general hospital; excellent salary, one month vacation after a year's service. Apply, Personnel Director, Good Samaritan Hospital, Cincinnati 20, Ohio.

ANESTHETISTS: Charleroi-Monessen Hospital (general, 238 beds and 36 bassinets), North Charleroi, Pa. (HU 3-5561). Salary open plus maintenance, sick leave, holidays, month paid vacation annually, etc. Call or write administrator.

WANTED: NURSE ANESTHETIST—A.A.N.A. membership. Excellent salary and opportunities. Paid vacation. Excellent working conditions. Rotating call every sixth night and sixth weekend. Day off following call. 550 bed hospital. Staff of eight anesthetists. Apartment and full maintenance available. Apply: Miss Mildred Hodges, Chief Anesthetist, Missouri Baptist Hospital, 919 N. Taylor Ave., St. Louis, Mo.

NURSE ANESTHETISTS —FLORIDA! Take advantage of Florida's sunshine and pleasant living. Jacksonville's rapid growth has created a need for hospital expansion. St. Vincent's has just completed an addition of 150 beds and has completely remodeled the existing hospital, which is located on the beautiful St. Johns River. Need 4 qualified anesthetists at once to complete a staff of 9. Beginning salary $450.00 per month with merit rating increases. Call rotation every 4 nights with time off following calls. 2 weeks annual vacation with graded increase to 4 weeks after 5 years service. 2 weeks sick leave. New nurses home under construction. No obstetrical call for surgical anesthetists, but obstetrical anesthetists are needed also, so applications are in order for either position. Only emergency operations on Saturday or Sunday. Apply: St. Vincent's Hospital, Jacksonville, Florida.

NURSE ANESTHETIST WANTED: For obstetrical anesthesia in department averaging 250-300 deliveries monthly. Salary $400 month plus complete maintenance, including room, meals, phone and laundry. Vacation and sick time granted. Apply: Peoples Hospital, Akron, Ohio.

NURSE ANESTHETIST: Salary $450 per month. No call. Write: Director of Anesthesiology, Zieger Osteopathic Hospital, 4244 Livernois, Detroit 10, Michigan.

NURSE ANESTHETISTS: Two needed at a small approved general hospital in Pittsburgh. Supervised by a qualified anesthesiologist. Salary open. 35 and under preferred. Apply: Louise Ludewig, Superintendent, St. Margaret Memorial Hospital, 46th Street, Pittsburgh 1, Penna.

ANESTHETIST: Obstetrical anesthesia only. 40-hour week. Pleasant working conditions in modern obstetrical department. Hospital located in a beautiful lake surrounded university city. Liberal vacation, sick leave, social security, group life and hospital insurance, pension plan. Apply: W. D. Barclay, Assistant Administrator, Madison General Hospital, Madison, Wisc.

ATTRACTIVE POSITION FOR NURSE ANESTHETIST: Approved hospital in Denver, Colorado; 198 beds. Will be one of 5 anesthetists. Good salary and working conditions with sick benefits, vacation and holidays. Apply: Personnel Office, Presbyterian Hospital, 19th at Gilpin St., Denver, Colorado.

TWO NURSE ANESTHETISTS: Approved 143-bed general hospital, Bristol, Tenn. 44-hour week, alternate call between 3 anesthetists. New, modern hospital, pleasant working conditions. Salary $400 per month with $25 increase at end of six months. Vacation and two weeks sick leave annually. Laundry, meals, and room in new nurses home. Write: Marigold Jones, Dept. of Anesthesia, Bristol Memorial Hospital, Bristol, Tenn.

WANTED: Graduate nurse anesthetist willing to assist in nursing capacity and give light general anesthesia when necessary. Excellent hospital facilities. Five and a half day week. Salary open. Large eastern city. Apply: Dr. Joseph Michael Tighe, 6601 York Road, Baltimore 12, Maryland.

NURSE ANESTHETIST: 5-day, 40-hour week. 73-bed general hospital expanding to 120 beds. New air conditioned surgery will open in January. Excellent pay depending on experience. All benefits including pension. Maintenance provided. Call or write: Administrator, Community Hospital, Geneva, Illinois.

ANESTHETIST - NURSE: 250 bed general hospital; excellent working conditions and personnel policies. Write: Mr. Bart Stajich, Assistant Administrator, Columbia Hospital, 3321 N. Maryland Avenue, Milwaukee 11, Wisconsin.

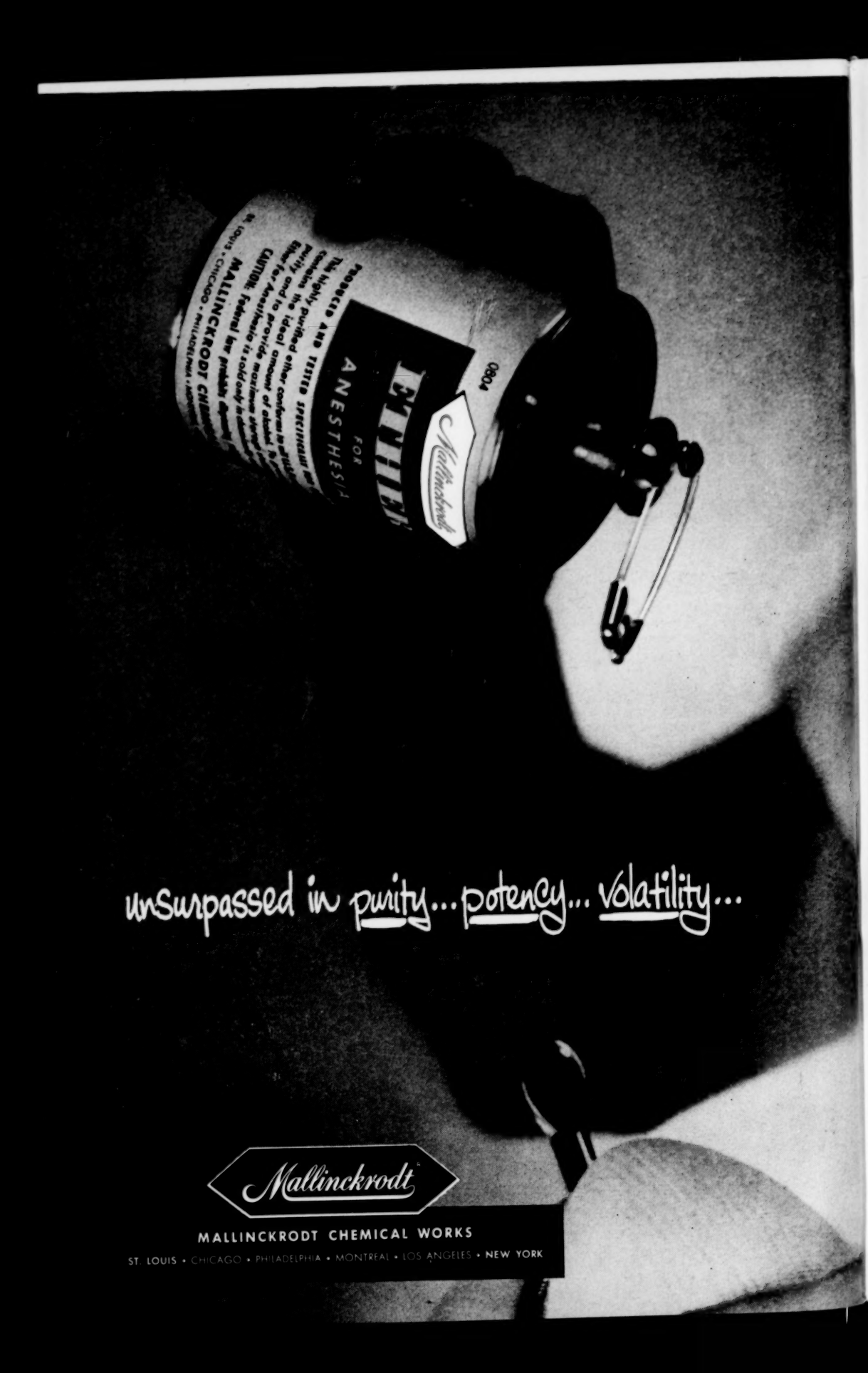
TWO NURSE ANESTHETISTS: for a 200-bed general hospital. Salary $500 per month. Apply C. K. Shiro, Administrator, Montana Deaconess Hospital, Great Falls, Mont.

Positions available for two Nurse Anesthetists. Salary, $400.00 per month. 441-bed General Hospital. Hamot Hospital, Erie, Pa. Contact the Director for full information.

THE TWENTY-THIRD QUALIFYING EXAMINATION

Will be Conducted on Saturday, May 12, 1956.

The deadline for accepting completed applications including the transcripts is April 2. Notice of eligibility will be mailed about April 9.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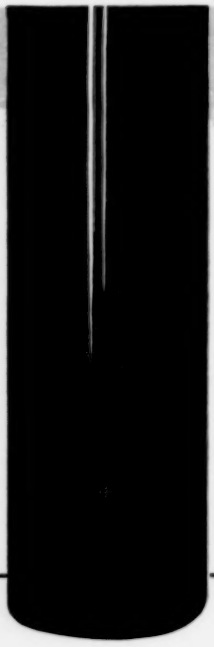

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